Volume 13, Number 2
FEBRUARY 1953

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"Cardiac Asthma"

JONATHAN C. MEAKINS*

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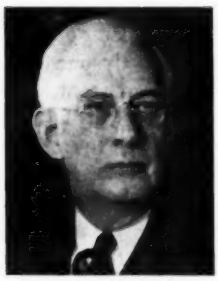
The following address briefly outlines the basic differences between traditional bronchial asthma and so-called "cardiac asthma." It attempts to show that the fundamental differences have been confused by the use of the common word "asthma." Further, these differences are of conspicuous importance, not only as to etiology, but also as to prognosis and therapy.



J. C. MEAKINS

THE term "cardiac asthma" should be replaced in the medical nomenclature by "paroxysmal cardiac dyspnea." The tremendous number of adjectives associated with the word "asthma" make the condition very difficult to define. From a puristic point of view we should restrict its use to that respiratory disturbance of function called bronchial asthma, for when we continue to use it for all and sundry types of dyspnea we get into a bog of contradictions. Perhaps it would be well to clarify the term bronchial asthma and then to differentiate or concretely describe the process so commonly called by what I firmly believe to be a misnomer—cardiac asthma.

Bronchial Asthma

Bronchial asthma may occur at any age. The duration of an attack may vary from a few minutes to many hours. The duration of the disease may be a lifetime, and the condition frequently is influenced by emotional and environmental factors. Between attacks there is little or no exertional dyspnea unless there are concomitant or associated pulmonary lesions such as diffuse bronchiectasis, emphysema or pulmonary fibrosis due to a variety of causative agents, among these being beryllium, which these days is becoming increasingly important in this regard.

The clinical signs of bronchial asthma are clearcut. The sputum is scanty and tenacious, and contains mucus plugs and Curschmann's spirals. The eosinophils and the Charcot-Leyden crystals are almost diagnostic, and when there is secondary infection from the accessory sinuses of the nasal passages or resulting from a previous attack of viral or bacterial pneumonia, the mucopurulent sputum is quite distinctive.

Dry rales are distributed throughout the lung fields in a patchy fashion; moist rales seldom are heard except in the presence of a secondary infection.

Much confusion has arisen concerning the heart and circulation in bronchial asthma. An interesting association which frequently is seen in a large clinical practice is that of bronchial asthma of the pure type and paroxysmal or transient hypertension. These may occur concomitantly, but very often they occur independently as if both were to some degree the result of a common emotional factor. As a consequence of this association, attempts have been made to prove that electrocardiographic changes may be associated with asthma—that is, with bronchial asthma. There is no foundation for such a concept. While it is true that on occasion transitory right axis

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

deviation may be detected during an asthmatic attack, this is undoubtedly positional and is not inherently an abnormal cardiac deviation.

During an attack of bronchial asthma the pulmonary arterial pressure is increased during expiration and falls suddenly at the end of expiration. Some attempts have been made to link this finding with right-sided heart failure; however, further analysis will reveal that other factors are present if right-sided heart failure occurs in addition to asthma. Furthermore, patients with bronchial asthma have normal circulation times. Frequently, due to associated or additional causes, cor pulmonale—that glib term which sounds so mighty—is in reality right-sided heart failure and is not a part of bronchial asthma. Associated conditions which may cause this condition, especially in later life, are emphysema and/or widespread fibrosis secondary to some other factor—and in neither of these is there any evidence of pulmonary edema.

"Cardiac Asthma"

With that background, let us discuss that misnomer, "cardiac asthma," which is not asthma at all but a paroxysmal type of dyspnea. Although it may be nocturnal in character it is more likely to be related to physical exertion, the latter in such cases always being accompanied by dyspnea between paroxysms. Thus it differs from pure bronchial asthma, in which the dyspnea does not occur in the free periods unless there is some associated respiratory disturbance.

A fundamental requirement for paroxysmal cardiac dyspnea, as I shall call it, is an increase in pulmonary venous pressure. Without this, paroxysmal dyspnea cannot occur, and this increased pressure is the keystone to our concept and eventually to the treatment.

There may or may not be a conspicuous increase in pulmonary arterial pressure. If arterial pressure is increased, it is an exaggeration of the normal in the opposite direction to that in bronchial asthma; the rise occurs with inspiration and the fall with expiration, whereas in bronchial asthma the pressure rises steeply with expiration and falls with inspiration.

The increased pulmonary venous pressure leads to a series of local pulmonary disturbances, including pulmonary venous engorgement, alveolar edema and pulmonary rigidity, as a result of which certain portions of the lungs become like erectile tissue. Another resulting disturbance is swelling of the bronchial mucous membrane, adding to the circulatory disturbance a certain degree of obstructive inspiratory and expiratory embarrassment. This, however, is minor compared with the turgescence resulting from increased pulmonary venous pressure and edema.

As a result of these circulatory disturbances there is dyspnea on exertion, in some cases almost constant, which is quite different from the dyspnea of bronchial asthma. The cough of cardiac dyspnea is conspicuously different from that of bronchial dyspnea. The sputum in bronchial asthma is tenacious and scanty; that in cardiac dyspnea is fluid and abundant, and may be increased to conspicuous amounts of serous, blood-tinged or even bright red, thin edema fluid.

Paroxysmal cardiac dyspnea may occur during sleep and therefore is often confused with bronchial asthma which, if caused by feathers, wool, horse hair or other epidermal agents, occurs most often during sleep, but the clinical pattern is entirely different. The respirations in cardiac dyspnea are rapid and shallow and not obstructive in any sense of the word; the rales are moist and located at the bases of the lungs and not in the upper lobes except in very extreme situations, and are caused by edema and not by bronchial obstruction or mucus secretion. Dry rales are relatively rare, and when present in paroxysmal cardiac dyspnea they may be and probably are due to edema of the bronchial mucous membrane. There is no evidence of emphysema. Roentgenograms reveal bilateral engorgement in the lower portions of the lungs. There is a reduced vital capacity at all times and not just during a paroxysm. The circulation rate is prolonged, there is acute arterial anoxia, and an acute carbon dioxide and lactic acid acidosis is present; all these manifestations conspire to make the dyspnea so much more agonizing to the victim.

Under what conditions do we find paroxysmal cardiac dyspnea? The first—and probably the most important, because it occurs early in life—is mitral valvular disease, particularly mitral stenosis. The degree of dyspnea and of the paroxysms both on exercise and during sleep is a good indication of the severity of mitral stenosis. The next most common cause is hypertension with left-sided cardiac failure. Associated from a similar point of view in the dynamics of this circulatory disturbance is aortic stenosis, which is really a form of hypertension with the block occurring at the aortic orifice instead of at the peripheral arterioles. A fourth cause is left ventricular failure due to myocardial infarction. These conditions account for more than 95 per cent of cases of paroxysmal cardiac dyspnea.

One does not need an electrocardiograph to indicate the treatment required in a case of paroxysmal cardiac dyspnea; one needs only a keen sense of medicine at the bedside.

Whenever there is paroxysmal cardiac dyspnea there is always an actual or incipient degree of pulmonary edema. But why does it occur under certain specific conditions? The reason for its appearance following exertion is obvious, but why does it occur during sleep?

An interesting fact about respiration is that an ordinary man does not like to have his respiratory movements, either thoracic or abdominal, impaired in the recumbent position. Women, however, don't mind it at all, and probably that explains their favoritism for thoracic breathing during those periods of respiratory embarrassment in the eighth and ninth months of pregnancy. I have often wondered what would happen to a man in the same circumstances; I am afraid he would be a most embarrassed individual—I don't mean sartorially, but respiratorially.

An example of this is a man with lobar pneumonia, although such cases seldom are seen today. That man always wanted to sit up; it wasn't true orthopnea; it was just that he breathed more easily in that position.

There is also the matter of a slowly accumulating anoxia. This may be due to increased abdominal pressure from a large liver or to ascites, or to a combination of the two. The patient slips down on his pillow, his respiration becomes slowly embarrassed, and in the course of time he awakes in terror—and that terror is of suffocation. How are we going to protect this individual from these attacks and assist him when he has them? By the time the doctor reaches his home he has recovered from the attack and wants either to go back to sleep or to sit in his favorite arm chair the rest of the night. A sedative on retiring helps. Morphine is contraindicated in acute cases of bronchial asthma, but as James Mackenzie pointed out almost 60 years ago a hypodermic injection of morphine may give relief for a whole night in the cardiac case, and sometimes he seemed to think it would have a delayed action over the day and night. I often queried him on this, but he never gave me an answer. There is, however, a hazard in the long-standing cases that morphine may become a two-edged sword.

Whereas ADRENALIN® and epinephrine are indicated in bronchial asthma, they make cardiac dyspnea much worse. Aminophylline is useful occasionally in bronchial asthma; this is true also in paroxysmal cardiac dyspnea, but one wonders how much of the good effect is due to its action as a vasodilator and how much is due to its action as a bronchial dilator. If it is a vasodilator, it is not indicated in cases of mitral stenosis.

If you have a patient who is subject to these nocturnal attacks, I would advise that you have your patient invest in a small tank of oxygen and a simple mask. Then if he awakens in the night in terror of suffocation, he can turn over, put on the mask and turn on the oxygen, and get relief.

All patients suffering with paroxysmal cardiac dyspnea should be on maintenance doses of digitalis, and by that I do not mean one of the high-powered new glycosides which probably are the most common cause of intoxication among admissions to the medical ward of any general hospital. The swing is away from these specific glycosides and the trend is toward the Withering and dry digitalis leaf prepared under proper pharmaceutical standards. It is simple and safe; overdosage is difficult.

The Changing Pattern of Pulmonary Disease

HAROLD L. ISRAEL*

University of Pennsylvania Graduate Hospital, Philadelphia

The major pulmonary diseases of the past generation have waned in importance and diseases that were rarities or unrecognized 35 years ago are major problems at the present time. Continued epidemiologic study is desirable to impress both the clinician and the pathologist with the diminishing frequency of bacterial infections and the increasing prevalence of viral, fungal, occupational and degenerative pulmonary diseases.

THE first edition of Norris and Landis' text on pulmonary disease appearing in 1917 devoted twice the number of pages to tuberculosis and pneumonia as it did to bronchiectasis, abscess, fungal infections, embolism and infarction, pneumoconiosis, fibrosis, emphysema and carcinoma combined. There is no mention in the index of viral or rickettsial diseases nor of coccidioidomycosis or histoplasmosis. The epidemiologic pattern of respiratory tract disease has changed profoundly since 1917. The continued decline in tuberculosis has increased the relative frequency and an aging population has increased the absolute frequency of the nontuberculous pulmonary diseases. Viral and fungal infections and degenerative disorders now represent major problems in pulmonary disease, and familiarity with their natural history is essential for the clinician.

To some extent the changing emphasis represents advances in the understanding of disease. Histoplasmosis formerly may have been as prevalent in Missouri as it is today. To a degree the changes reflect such brilliant developments in therapy as the antibiotics. Major credit for the changing picture cannot be given medical science, however, for it is well known that the death rate from tuberculosis began its steady decline years before the tubercle bacillus was found and long before the introduction of the real and fancied therapeutic advances which have been applied in the past half century. In a similar fashion pneumococcal lobar pneumonia declined markedly in severity before the relatively recent introduction of serums, sulfonamides and antibiotics.

The diminishing fatality rates from these bacterial diseases of early adult life have permitted the survival of an older population with a different array of respiratory disorders, notably, degenerative diseases such as emphysema and neoplasms. Newer methods of transportation and the greater mobility of our population in time of war have had a profound effect in accelerating the spread of viral infections. Coccidioidomycosis might still be

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a benign curiosity in a few isolated desert areas of Arizona and California—the influx into these areas of previously unexposed workers and soldiers resulted in many thousands of clinically significant infections.

H. L. ISRAEL

With marked changes going on in the human population as well as in its microbial parasites, it is uncertain whether or not rickettsial and viral pneumonias were prevalent but unrecognized in 1917. These organisms may have assumed significant roles as pathogens only since bacterial infection diminished in prevalence. A plausible explanation is that the pathogenicity of bacteria having declined as the result of environmental and constitutional changes in the human host, the diminished competition of the bacteria has permitted viruses to flourish. Burnett[1] has proposed the interesting hypothesis that viruses represent the degenerate descendants of larger pathogenic forms which have undergone an evolutionary sequence from bacteria to *Rickettsia* to larger viruses to smaller. At each step one or more of the enzyme systems needed for synthesis has been discarded so that increasing fastidiousness in regard to requirements for growth in artificial mediums is shown and artificial culture becomes impossible. Eventually the virus becomes no more than a large nucleoprotein molecule which has lost all properties but the capacity to reproduce and to abstract from the cell the material and energy needed for multiplication.

The diseases whose epidemiologic patterns will be briefly reviewed include such common but diverse illnesses as the common cold, histoplasmosis, thromboembolism and bronchogenic carcinoma, as well as less common but instructive diseases such as epidemic pleurodynia, Q fever, coccidioidomycosis, sarcoidosis and beryllium granulomatosis.

Viral Infections

An interesting application of the household study has been instituted by Dingle and his associates[2] in Cleveland. A group of stable families has been enrolled in an intensive study including weekly throat cultures, periodic serologic tests and careful medical and laboratory study of every illness, however minor. A preliminary report has been made of a year's experience in which there were 61,070 person-days of observation and 1,391 illnesses, representing 6.22 illnesses per person per year. Approximately 75 per cent of all the illness in these households was due to respiratory disease. A careful clinical and bacteriologic study of the 1,040 respiratory illnesses resulted in diagnoses of bacterial infections in but 1.5 per cent—2 cases of pneumococcal pneumonia and 14 cases of streptococcal pharyngitis. The nonbacterial infections were classified as follows: atypical pneumonia, 7 cases; pharyngitis and tonsillitis, 42 cases; common cold, 420 cases; other respiratory diseases, 555 cases. These astonishing figures emphasize the present importance of viral respiratory disease.

The common viral infections of the respiratory tract are at present classified as the common cold, pharyngitis, acute respiratory disease, influenza and primary atypical pneumonia. Clinically it may be impossible to distinguish these illnesses one from another and specific diagnostic tests are available only in the last two; hence, epidemiologic studies have been able to contribute relatively little to our knowledge of these infections. Much of our knowledge has come from the experimental investigations of the Army Epidemiologic Board in World War II and the Common Cold Research Unit in England.[3] From such studies we have learned that the common cold has an incubation period of from two to three days, yields brief immunity and is rarely accompanied by fever. The virus is demonstrable in the nasal secretions 24 hours before the onset of symptoms and may persist for a week.

Acute respiratory disease (ARD) resembles the cold but has a longer incubation period and fever is usually present. Viral pharyngitis can simulate acute streptococcal pharyngitis or tonsillitis so that differentia-

tion is impossible without bacteriologic study. Influenza epidemics have not been of great severity in recent years. Four strains, A, A′, B and C have now been isolated, which explains the difficulty of immunization. Influenza has been infrequent in Philadelphia except during epidemic outbreaks. Viral pneumonia is accompanied by formation of cold agglutinins in 50 to 60 per cent of the patients; it is possible that those developing cold agglutinins represent one entity, while those that do not form another group. This question will presumably remain unanswered until isolation is achieved of the virus or viruses responsible for atypical pneumonia.

Psittacosis is frequently classified as a viral disease, although the micro-organism is so large that it is classed by others as a subgroup of *Rickettsia*. A wide variety of birds and fowl can be host to viruses of this group. As a rule infection occurs only from a sick bird to man; although Meyer and Eddie[4] observed a patient whose sputum contained the virus eight years after an attack of psittacosis, no secondary cases were observed among contacts of this man. On the other hand, some strains appear to become adapted to man and are highly contagious, as in the 1940 epidemic in Louisiana.

Strong evidence has been accumulated recently to indicate that Coxsackie virus, which was originally recovered from cases resembling poliomyelitis, is the agent for the illness which has long been known as Bornholm disease—epidemic pleurodynia.[5] During the summer of 1951 numerous patients were seen with severe chest pain simulating pleurisy or myocardial infarction; these may have been instances of Coxsackie virus infection but the clinical diagnosis is most difficult in the sporadic case. Serologic diagnosis is not at present reliable but the virus can be isolated from the throat or stools.

Rickettsial Infections

Q fever was first described as a rickettsial infection in 1937; it has since been found to be worldwide in occurrence. It is especially prevalent in southeastern Europe where it has been familiar as the Balkan grippe. Recently it has been demonstrated to be endemic in southern California where it is estimated that 50,000 cases—for the most part mild or subclinical—have occurred in the past few years. Cows, sheep and goats are infected in this area; infection occurs through ingestion of raw milk or meat, or may be airborne. Two cases have been detected in Philadelphia, both in workers in wool-processing plants. Serologic study of the other workers in one of these plants disclosed evidence of Q fever infection in 65 men, in 35 of whom the infection had been subclinical.[6]

Bacterial Pneumonia

There is growing evidence that the bacterial pneumonias are not actually primary infections. Hodges and MacLeod's observations at army installations where pneumococcal pneumonia was epidemic indicated that, with high *Pneumococcus* carrier rates, outbreaks of pneumococcal pneumonia were correlated with epidemic surges of viral infection.[7] More recently, influenza antibodies have been demonstrated[8] in the majority of a group of patients who had bacterial pneumonia. It is probable that viral infection of the bronchial tree or actual viral pneumonia is the underlying process preceding bacterial pneumonia, and that the nature of the latter process is determined by the predominant flora carried in the respiratory tract.

There has been a marked decline in the mortality from lobar pneumonia in this country between 1925 and 1937, with case fatality rates falling to the lower levels that had characterized pneumococcal pneumonia in England and western Europe. Since the introduction of sulfonamides and the antibiotics, case fatality rates in patients treated in large American municipal hospitals have further declined to 5 per cent.[9] It should be recognized that the mortality is considerably lower among young adults: Rates of less than 1 per cent were reported in the army and navy during the last war. The higher death rate in hospitals is accounted for by the continued high mortality among elderly patients and alcoholics.

Relatively little information is now avail-

able concerning the morbidity from pneumonia since the general use of antibiotics has reduced the need for hospitalization of patients with pneumonia; in hospitals bacteriologic study of pneumonia cases now receives little attention. It is certain that bacterial pneumonias are less often encountered in hospitals and it is probable that they are less often seen at home. Pneumonias due to gram-negative organisms appear to be increasing in frequency, presumably because suppression of the cocci by penicillin has facilitated growth of penicillin-resistant organisms.

Bronchiectasis

This common but often unrecognized disease presents an excellent example of the need for epidemiologic study of a noncommunicable but nonetheless preventable disorder. It is clear that bronchiectasis may be pathologically the result of a variety of factors, congenital or acquired, damaging the bronchi or lungs; foreign bodies, childhood pneumonia, tuberculosis and lung abscess are among the more common causes. Advances in prevention and treatment of these diseases may materially reduce the future prevalence of bronchiectasis. It is well known that the pneumonias accompanying the viral infections of childhood, for example, measles, are frequent precursors of bronchiectasis, but it is not established whether the viral infection alone or the superimposed bacterial infection is responsible; hence it remains to be demonstrated whether control of the bacterial element by antibiotic therapy will prevent the development of bronchiectasis. Attention has recently been directed to the frequency with which bronchiectasis is associated with congenital anomalies such as situs inversus; the increase in viscosity of pulmonary secretions associated with congenital cystic fibrosis of the pancreas has also been shown to be an important cause of bronchiectasis.

It is remarkable how little is known of the course and prognosis of bronchiectasis. Marked difference of opinion exists as to whether bronchiectasis is a "progressive" disease—whether the damage if not extirpated can spread to involve other lobes. The affirmative is often argued by thoracic surgeons who have also urged resection on the basis of the high mortality[10] observed in untreated patients. The statement[11] that few patients with bronchiectasis survive beyond the age of 40 is often quoted. It should be recognized that such observations were made on patients diagnosed years ago when bronchiectasis was considered to be characterized by profuse purulent expectoration and clubbed fingers and when bronchograms were rarely secured in the absence of advanced symptoms. At present, it is the practice to employ bronchography in many patients not chronically ill who have recurrent pneumonias, chest pain or hemoptysis; this has demonstrated that bronchiectasis is far more common than previously thought.

The proportion of patients with bronchiectasis who eventually develop chronic septic infection is by no means established and the current practice of recommending surgical excision in all cases of bronchiectasis is not based on factual data secured from prolonged observation of patients with bronchiectasis. At present such information would be most difficult to acquire since recent papers deal only with results of cases treated surgically.

Accurate knowledge concerning the development and course of bronchiectasis would best be obtained by application of a case-finding technic similar to that employed in tuberculosis, namely, by securing bronchograms of large numbers of normal controls in various age groups and by repeating these studies at 5 or 10 year intervals in order to detect the development of bronchiectasis. We have given consideration to such a study but have been deterred by the fact that bronchography is not an innocuous procedure; it is doubtful whether the introduction of LIPIODOL® in normal controls is justifiable. Recently watermiscible compounds have been developed which are rapidly cleared from the lungs and apparently leave no residue. If these compounds are demonstrated to be innocuous it may be possible to obtain bronchograms of normal persons on a large scale and thereby secure much needed information about the pathogenesis of bronchiectasis.

Fungous Diseases

For many years it has been the custom of physicians and roentgenologists to take refuge in the suggestion of fungal infection whenever a particularly exotic or baffling pulmonary infiltration is encountered. Rarely in this area have such lesions proved to be of fungal etiology; if *Monilia* or *Aspergilli* have been demonstrable in the sputum, they have usually proved to be secondary invaders. Within the past few years, however, two fungal diseases of national importance have been identified and thoroughly studied. The investigations of these infections—coccidioidomycosis and histoplasmosis—have been models of epidemiologic study. The acquisition of our knowledge of these two diseases has followed a familiar pattern, both having been described originally as rare but universally fatal infections; only later was it demonstrated that the fatal cases represented an unusual manifestation of a common and usually benign infection.

Coccidioidomycosis was first recognized in the form of the fatal coccidioidal granuloma and was shown to be a fungal infection in 1900. It was not until 1937 that this organism, *Coccidioides immitis*, also was shown to be the cause of the mild respiratory infection associated with erythema nodosum that was known as "valley fever" in the San Joaquin Valley of California. A few years later, Aronson, studying tuberculosis among the Indians in Arizona, noted that in many subjects with pulmonary calcifications the tuberculin tests were negative. Application of the coccidioidin skin test demonstrated a high frequency of reactions and he suggested that coccidioidomycosis was responsible for the calcifications.[12]

Our knowledge of coccidioidomycosis grew rapidly during the war years when large numbers of men were stationed in the southwestern desert areas of this country for training. Erythema nodosum and varied pulmonary manifestations became a serious problem. The studies by Dr. Charles Smith of Stanford University and his associates demonstrated that persons entering these dusty areas were rapidly infected.[13] Erythema nodosum developed in approximately 5 per cent, and acute respiratory illness with roentgenologic study usually showing a transient pneumonitis was observed in 25 per cent. In many instances resolution of the pneumonic infiltration left residual nodules or thin-walled cavities and these lesions, which mimicked tuberculosis and neoplasm, presented a major problem in diagnosis in military medicine. The majority of these chronic residuals have been benign but a few have shown local progression and have required surgical resection. Instances of malignant generalized coccidioidomycosis progressing rapidly from the initial infection were encountered in 0.25 per cent of white soldiers infected and in 3.4 per cent of all others.

It is to be noted that spread of coccidioidomycosis from one person to another has not been demonstrated and it appears that infection is possible only by inhalation of dust containing the spores. For all practical purposes this disease can be excluded in persons who have never resided in or visited the desert areas of Arizona, California and neighboring states. There have been rare exceptions to this rule such, for example, as was provided by the outbreak of coccidioidomycosis in an army unit in Georgia comprised chiefly of recruits who had entered the service from the eastern states. Investigation indicated that a few members of the unit had come from desert training areas with dust-laden blankets and duffel bags which were responsible for infection of the other members of the unit.

Our knowledge of the epidemiology of histoplasmosis is of even more recent origin. A review of this disease published in 1945 reported less than a hundred known cases, practically all fatal. A few years earlier the Public Health Service investigations of tuberculosis had revealed pulmonary calcifications, regarded to be of tuberculous origin, in large numbers of children and adults who failed to react to tuberculin. This led at first to the erroneous conclusion that x-ray examination was far superior to the tuberculin test for the detection of tuberculosis. It was pointed out that the calcifications were numerous in Ten-

nessee where the tuberculosis death rate was relatively low, whereas an area in Alabama with a higher tuberculosis death rate showed a considerably lower incidence of the pulmonary calcifications. Considerable study was devoted to the possibility that differences in the mineral content of water and food might be responsible for the varying prevalence of calcifications.

Influenced by Aronson's observations on the frequency with which Arizona Indians with calcifications reacted to coccidioidin, and at Smith's suggestion, the skin reaction to histoplasmin was studied and this proved the key to the puzzle. Positive histoplasmin tests proved more frequent than tuberculin reactions in patients with pulmonary calcifications in Ohio and Missouri valley states. The subsequent studies by Furcolow and associates of the Public Health Service completed the proof of the role of *Histoplasma* in producing pulmonary calcifications. Children have been detected with widespread pulmonary infiltrations, *H. capsulatum* cultured from their secretions, and the lesions observed to undergo calcification.[14] Skin tests with histoplasmin indicate a frequency of infection ranging from 90 per cent in young adults in Missouri to approximately 10 per cent in the Atlantic and Pacific coastal states. Among the histoplasmin reactors in the latter states are many persons who have not visited the endemic areas; from this evidence and from study of fatal cases, it is clear that infection with *Histoplasma* is not restricted to any area of this country. It is also evident that in such states as Missouri and Kentucky infection is almost universal and, with few exceptions, benign. Intermediate between this type of infection and the rather rare fatal systemic infections is the last type to be described: chronic disseminated histoplasmosis which may cause illness of several years' duration with predilection for involvement of the larynx, adrenal glands and lungs and which, both clinically and histologically, may simulate sarcoidosis.[15]

Sarcoidosis

It is only 15 years since attention was first directed in this country to sarcoidosis as a systemic disease. Once the syndrome was recognized, sarcoidosis proved to be a relatively common disease. It remains a disease of undetermined etiology. Scandinavian research workers[16] have reported the isolation of a virus from 5 cases of sarcoidosis but have not succeeded in establishing this virus as the cause of disease. Some experienced physicians still consider sarcoidosis an atypical form of tuberculosis, but the intensive investigation of recent years has failed to yield substantial evidence for this theory and much indirect evidence has been accumulated which argues against a tuberculous etiology.

An observation of great interest was made by the Army Epidemiologic Board in reviewing the data on sarcoidosis detected among army personnel during the last war. It was noted that sarcoidosis was disproportionately frequent both in Negroes and whites inducted in the southeastern states. This observation has been extended by Michael and his associates[17] who found a record of the birthplace of 226 of the army patients. The rate in southern Negroes was 15 times that of northern-born Negroes; the rate in southern whites was 6 times that of northern-born whites and was higher than that of northern Negroes. This remarkable geographic distribution would appear an invaluable clue in the search for the cause or causes of sarcoidosis. It suggests either an infectious agent, prevalent chiefly in this country in the rural south, or some constitutional or environmental abnormality, occurring in childhood years, resulting in a modification of response to irritants or infection in later years. In any event no theory of the etiology of sarcoidosis can be seriously entertained that does not agree with these geographic facts.

The Scandinavian use of the term "benign lymphogranulomatosis" for this disease, and some of the earlier American reports on sarcoidosis have given a falsely sanguine impression about its course. At one time, except for the risk that tuberculosis would develop, sarcoidosis almost invariably was considered to be benign. A total of 59 patients with sarcoidosis have been followed in our clinic for an interval of more than four years. There

have been 7 deaths—a gross mortality of 12 per cent. One death was from tuberculosis; the others were due to respiratory insufficiency resulting from sarcoidosis. It is clear that superinfection with tuberculosis is less frequent than has been reported and that sarcoidosis is in its own right much more malignant than has been recognized.[18]

Pulmonary Embolism and Infarction

The diminishing importance of infection as a cause of postoperative morbidity and mortality has increased the relative importance of thromboembolic disease, and as a result great attention has been given the prevention of venous thrombosis by employment of early ambulation, prophylactic femoral vein ligations and the use of anticoagulants. It is therefore disappointing that recent studies indicate that no reduction in frequency of thromboembolism has been achieved. The explanation for this failure lies in the fact that the factors which predispose to venous thrombosis—besides immobilization in bed—are age, obesity, anemia, heart disease, neoplasm and extensive surgical procedures. It is evident that, with an aging population and with more frequent hospitalization for radical surgical therapy of patients with neoplasm often complicated by anemia and heart disease, an increased danger of thromboembolism must be anticipated.

Although pulmonary embolism and infarction have received the greatest attention as postoperative problems, it should be recognized that their frequency is actually greater among medical patients and is by no means negligible in ambulatory patients. As the control of infection by antibiotics improves, the likelihood that an apparent pneumonia or pleural effusion is of thrombotic rather than infectious etiology increases; there is need for more frequent consideration of thromboembolic disease in the diagnosis of both acute and chronic pulmonary disorders.

Pulmonary Emphysema and Fibrosis

Brilliant advances have been made in recent years in elucidation of the physiologic disturbances which result from emphysema and fibrosis; unfortunately, relatively little has been learned of their incidence and genesis. One significant advance in our knowledge of this group of diseases has been the recognition of beryllium granulomatosis.[19] Beryllium is an element which only recently has found extensive industrial use.

The first description of pulmonary disease in persons exposed to beryllium was in 1943 by investigators in Cleveland who observed in such workers acute and apparently benign pneumonitis. Between 1941 and 1943 three persons who had been employed in a Massachusetts fluorescent lamp factory were seen with severe respiratory symptoms and roentgenologic findings. First thought to be instances of sarcoidosis, these symptoms were astutely connected with exposure to beryllium. Subsequently hundreds of these cases have been recognized among industrial workers, research physicists, persons exposed to beryllium through breaking fluorescent tubes, and most remarkably among wives of industrial workers and among persons who merely resided in the neighborhood of plants using beryllium.

It is to be emphasized that beryllium is not a strongly toxic substance producing severe damage in all persons exposed; it appears that only a minority of those exposed develop the disease. As a result there is skepticism in some quarters as to the exact etiologic relationship of beryllium to this disease. The granulomatosis associated with beryllium exposure is a strange disease in many respects—the onset of symptoms is delayed for many years after the termination of exposure and severe respiratory insufficiency may precede manifest roentgenologic changes. Thus one young man who had had industrial contact with fumes from a beryllium alloy process in 1940 was subsequently inducted into the army and remained in excellent health until 1944, when respiratory symptoms developed. Chest roentgenograms were reported negative; by 1947 he was disabled by respiratory insufficiency and x-rays at that time revealed diffuse infiltration compatible with beryllium granulomatosis.[20]

The resemblance of this disease to sar-

coidosis is remarkable and the differentiation of the two may be impossible even with laboratory and histologic study. The bizarre epidemiologic features of beryllium granulomatosis provide food for thought. The development of extensive disease so many years after the termination of exposure to a noxious agent suggests that other diseases of unknown etiology may represent reactions to insults experienced years before and presents a striking challenge to the epidemiologist.

Bronchogenic Carcinoma

Intensive epidemiologic study is now in progress in an effort to explain the rapid increase in incidence of bronchogenic carcinoma. In 1912 Adler was able to collect only 374 cases of lung carcinoma from the world's literature; at the present time bronchogenic carcinoma is the most common neoplasm among men. Equally remarkable is the sex difference in incidence of pulmonary carcinoma; in several recent studies the ratios observed have approximated 20 males to 1 female.

Although occupational factors do not appear to influence the frequency of carcinoma of the lung, Wynder and Graham have noted that metal workers and painters comprise 16.7 per cent of the total number of patients with bronchogenic carcinoma.[21] The rapid rise among males has not been confined to industrial workers and attention has been directed to smoking as a possible cause. Two investigations have been reported, one in America and the other in England, with quite similar conclusions. Wynder and Graham found that 51.2 per cent of patients with lung cancer and only 19.1 per cent of controls had smoked in excess of a pack of cigarets daily for more than 20 years. Doll and Hill[22] noted that 4.2 per cent of a control group and 0.3 per cent of patients with cancer of the lung were nonsmokers; 26.0 per cent of men with cancer of the lung were heavy smokers compared to 13.5 per cent of the controls.

The increased prevalence of carcinoma among smokers appears confined to the epidermoid variety; the occurrence of adenocarcinoma of the lung bears no relation to smoking. Retrospective epidemiologic surveys of this type, in which smoking data are obtained by history taking, are not as convincing as planned observation studies. Studies of the latter type in which persons with various smoking habits will volunteer to remain under prolonged medical observation to determine the incidence of bronchogenic carcinoma are now being organized. Determination of the significance of smoking in the pathogenesis of bronchogenic carcinoma represents a real epidemiologic challenge and it is urgent that suitably large and prolonged studies be instituted in order to determine whether the incidence of this disease is influenced by tobacco in any form, moderate cigaret smoking or only by excessive cigaret smoking.

Other Diseases

As tuberculosis declines in frequency it may be anticipated that the value of the tuberculin test will further increase, for diagnostic as well as epidemiologic purposes. The use of the tuberculin test was in large measure responsible for the recognition of the importance of coccidioidomycosis and histoplasmosis. Recently study of tuberculin test data has demonstrated a significant geographic peculiarity in frequency of reactions to second strength tuberculin.[23] Tuberculin testing of student nurses in cities throughout the United States indicated a marked excess of second strength reactors in the south central states—most notable in Louisiana and surrounding states. This high frequency of weak reactors does not appear related to the prevalence of tuberculosis in this geographic area and appears best explained as a cross reaction to some infection localized to these states. Thus epidemiologic study with the tuberculin test appears to have delineated the geographic distribution of an infection of as yet completely unknown cause and manifestations! The success of epidemiologic analysis in elucidating the nature of coccidioidomycosis and histoplasmosis inspires confidence that further study will reveal a disease that will explain this interesting observation in the south central states.

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Postgraduate Clinical NOTES

1. BLEEDING GUMS

Several rare disorders of the blood or of the capillaries may give rise to bleeding gums. It is of the utmost importance to the patient that a correct diagnosis should be made early, and especially before any vigorous treatment is given. The principal diseases to be considered are acute leukemia, agranulocytosis, thrombocyptopenic purpura, scurvy, other hemorrhagic states, and occasionally chronic myelogenous leukemia. Agranulocytosis especially should be considered when the local condition fails to improve as soon as treatment is instituted. These other diagnoses should be considered if the patient looks ill or if additional clinical features such as purpura or anemia are evident. In such cases a full clinical examination should be made, including the family history and the patient's diet. It may be necessary also to investigate the total and differential leucocyte counts, the number of platelets, and the bleeding and clotting times. It is clear that in most cases such investigations are rendered unnecessary by clinical examination of the mouth and the general appearance of the patients.

Edward B. French, *The Practitioner*, September 1952, p. 300

Valvular Heart Disease in Older People

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Chronic valvular heart disease was found in 8.3 per cent of patients 50 years of age or older who were necropsied at the Mayo Clinic in 1950 and 1951. Although many lived to old age the valvular deformities presented hazards which indicate that antemortem recognition is of importance.

M. W. ANDERSON

DISEASE of the valves of the heart, although ordinarily considered to affect children and young adults, is by no means rare in older people. With the exception of occasional cases of syphilitic aortic insufficiency, a rare congenital malformation and those cases of calcific aortic stenosis in which etiology is controversial, rheumatic fever is the principal cause of valvular heart disease both in youth and in older patients.

Recently, Sprague and Carmichael[1] found rheumatic heart disease in 115 of 1,025 patients more than 50 years of age who were studied with necropsy. These authors expressed the opinion that rheumatic heart disease will be more common among older persons in the future as the result of improved treatment for rheumatic fever, congestive failure and bacterial endocarditis. In Clawson's[2] consecutive series of 30,265 necropsies there were 870 cases of rheumatic heart disease. This included 12 cases of acute rheumatic endocarditis, 15 with recurrent rheumatic endocarditis and 311 cases of chronic valvular deformities among persons 50 years of age or older. White and Jones,[3] who reviewed data on 3000 cardiac patients in 1928, found that 12.3 per cent of their patients with rheumatic heart disease were more than 50 years old. In still another study of 2000 consecutive necropsies by Wartman and Hellerstein,[4] there were 120 cases of rheumatic heart disease, in 49 (41 per cent) of which the patient had lived to be 45 years or older. Valvular heart disease in older people is not limited to chronic deformities. Typical clinical rheumatic fever and also histologic evidence for acute rheumatic carditis have been reported in patients more than 60 years of age.[5,6]

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

Procedure

To obtain information concerning valvular heart disease in older people who were observed both during life and with necropsy examination at the Mayo Clinic, the necropsy protocols, clinical records and gross pathologic specimens of all patients 50 years of age or older who died in 1950 and 1951 were

examined. Only those cases in which there appeared to be sufficient deformity to influence cardiac function were regarded as having valvular heart disease. This criterion eliminated from consideration hearts in which there were merely focal plaques of fibrous thickening and calcification without deformity of the leaflets, cases with calcification of the mitral ring without valvular distortion and some cases with minimal changes in the chordae tendineae.

Findings

General data (Table 1)—There were 896 consecutive necropsies, of which 554 were of male and 342 of female patients. Forty-six men (8.3 per cent) and 28 women (8.2 per cent) had valvular heart disease according to the definition of this paper. The highest incidence for both sexes was found from 70 through 79 years of age. The average age at death for all patients in this selected study with valvular heart disease was 69.3 years. There were 28 patients who died at 70 through 79 years of age and 12 who survived beyond 80 years. In 14 cases complete clinical data were lacking. This gave 60 patients 50 years of age or older who were studied both clinically and pathologically and 14 on whom only necropsy study was done. A cardiac murmur was detected in 47 and a clinical diagnosis of valvular heart disease was made in 36 (60 per cent) of the 60 patients. There were 17 patients with hypertension and also 17 with angina pectoris. Of the latter, 14 patients had aortic stenosis as the predominant valvular defect and 3 had predominantly mitral valvular deformity.

TABLE 1

INCIDENCE AND AGE OF DEATH IN PATIENTS 50 YEARS OF AGE OR OLDER DYING WITH VALVULAR HEART DISEASE

	MEN	WOMEN	TOTAL
Consecutive necropsy examinations	554	342	896
Number with valvular heart disease and per cent	46 (8.3)	28 (8.2)	74 (8.3)
Number of cases with valvular disease by decades			
50-59 years	8	8	16
60-69 years	16	2	18
70-79 years	16	12	28
80-89 years	6	6	12
Average age at death of patients with valvular disease, years	68.9	69.9	69.3
Age range, years	52-85	51-89	51-89

TABLE 2

TYPE AND INCIDENCE OF VALVULAR DEFORMITIES DEMONSTRATED AT NECROPSY IN 74 PATIENTS 50 YEARS OR OLDER

	MEN	WOMEN	TOTAL
Mitral valve deformity	15	18	33
Mild	13	15	28
Marked	2	3	5
Aortic valve deformity	29	12	41
Mild	20	7	27
Marked	9	5	14
Tricuspid valve deformity	0	3	3
Bacterial endocarditis	8	3	11
Acute	4	0	4
Subacute	4	3	7
Luetic aortic insufficiency	2	0	2
TOTAL	54	36	90*

*Total valvular deformities exceed the 74 cases because in a number of cases two or more valves were deformed.

Type of valvular deformity (Table 2)—In the 74 hearts a total of 90 significantly deformed cardiac valves were demonstrated at necropsy. Chronic mitral endocarditis was demonstrated in 33 (45 per cent) of the 74 hearts and aortic valvular disease of the calcific variety occurred in 41 cases (55 per cent). The tricuspid valve was involved in only 3 cases and there was no instance of pulmonary valvular disease. In 17 cases both aortic and mitral deformity was present. Pure aortic insufficiency due to luetic aortitis was found in 2 instances.

The incidence of mitral valve disease was significantly higher among female patients (18 or 64 per cent compared with 15 or 33

per cent in men), whereas calcific aortic endocarditis predominated in the men (29 or 63 per cent compared with 12 or 43 per cent in women). The 2 patients with luetic aortic disease were men and the 3 with rheumatic tricuspid deformity were all women.

When the degree of structural deformity as determined at necropsy was evaluated by arbitrarily dividing the aortic and mitral defects into those of mild and marked deformity, it was evident that the majority of the valvular defects were of the mild variety. Only 5 among 33 (15 per cent) of the hearts with mitral disease had marked mitral deformity and 14 of 41 (34 per cent) of those hearts with aortic deformity had severe lesions.

This expected finding of predominantly mild deformity, which was especially true in those patients with mitral valvular disease, must be strongly considered to be an important reason for the long survival. The average age of death was 62 years for the 5 patients with severe or marked mitral stenosis and was 66 years for 14 who had severe degrees of aortic stenosis. No patient with severe

FIGURE 1. The aortic valve from above, showing marked stenosis and calcification in a man of 83 years.

FIGURE 2. Moderate chronic rheumatic mitral endocarditis with stenosis and with dilatation of the left atrium found in an 83 year old woman.

TABLE 3

DATA CONCERNING 11 CASES OF BACTERIAL ENDOCARDITIS AMONG 74 CASES OF SIGNIFICANT VALVULAR HEART DISEASE IN PATIENTS 50 YEARS OR OLDER

AGE (YEARS)	SEX	VALVE INVOLVED	PRECIPITATING CAUSE
Subacute			
70	F	Mitral	None
77	M	Mitral	Prostatic resection
52	F	Aortic, mitral	Chronic glomerulonephritis
65	M	Aortic	Prostatic resection
57	F	Aortic	None
81	M	Mitral	Prostatic resection
52	M	Aortic	None
Acute			
54	M	Mitral	Diabetes, acute pyelonephritis
63	M	Aortic	Postoperative infection
62	M	Tricuspid	Postoperative infection
62	M	Aortic, tricuspid	Prostatic resection

mitral deformity exceeded 65 years whereas there were 7 with marked aortic stenosis who lived beyond 65 years of age and 4 were more than 75 years. Figure 1 illustrates marked aortic stenosis in a man who died at 83 years of calcific aortic stenosis with congestive heart failure. Long survival in a woman who lived to the age of 83 years with a moderate degree of mitral stenosis is illustrated in Figure 2.

Bacterial endocarditis—As indicated in Table 2 there were 11 cases with bacterial endocarditis of which 7 were subacute and 4 were of the acute or ulcerative type. The aortic and mitral valves were found to be the site for subacute bacterial endocarditis in older people, each in 3 instances, and in 1 case both valves were involved. The tricuspid and aortic valves were involved twice and the mitral once in the patients with acute bacterial endocarditis (Table 3). In 3 instances of subacute bacterial endocarditis and in 1 case of acute bacterial endocarditis transurethral prostatic resection had been performed within six months prior to death. Acute bacterial endocarditis was part of the picture of general sepsis which developed after laparotomy in 2 cases. In another case the patient was a diabetic man of 54 years who had acute pyelonephritis and whose involved mitral valve is shown in Figure 3. No predisposing cause for the infection was determined in 3 patients who died with subacute bacterial endocarditis. It is of interest that this complication developed and terminated fatally in 1 of the 2 patients who had luetic disease of the aortic valve with regurgitation, representing a rare coincidence of disease (Figure 4).

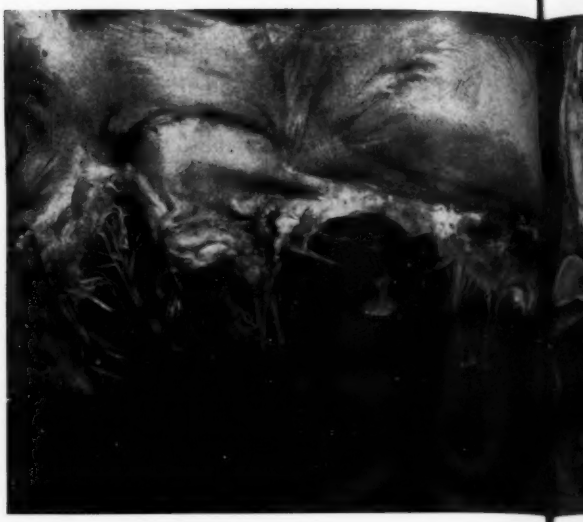

FIGURE 3. Acute bacterial endocarditis of the mitral valve in a man of 54 years who was diabetic and had acute pyelonephritis.

TABLE 4

CAUSE OF DEATH (74 CASES)

	DIAGNOSES
Congestive heart failure	30
Bacterial endocarditis	11
Cerebral vascular disease*	10
Coronary insufficiency†	9
Malignant neoplasm	9
Bronchopneumonia	6
Pulmonary embolus	6
Infarction of the intestines	3
Peritonitis (postoperative)	3
Postoperative shock	2
Acute pyelonephritis	2
Gastrointestinal hemorrhage	1
Arteriosclerotic gangrene	1
Renal infarction and uremia	1
Chronic glomerulonephritis	1
Pseudomembranous enteritis (postoperative)	1
Trauma	1
TOTAL	97‡

*Includes cerebral embolism and thrombosis, hemorrhage and infarction.

†Includes acute myocardial infarction and severe coronary insufficiency without myocardial infarction.

‡In a number of instances more than one cause was felt to contribute in causing death.

Immediate causes of death (Table 4)—In the 74 cases 97 pathologic diagnoses are included as significant factors in the cause of death, since in a number of cases two or more lethal lesions were active. Cardiac disability as manifested by acute or chronic congestive failure, coronary insufficiency and bacterial endocarditis was the principal cause of death.

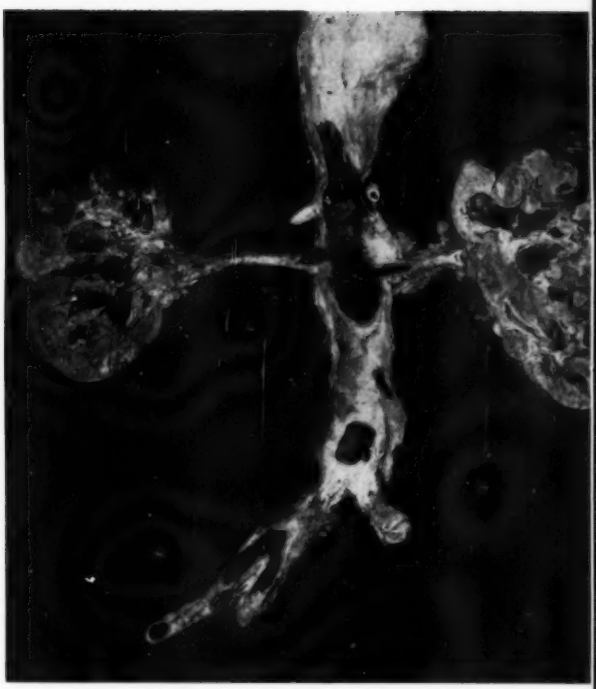

FIGURE 4 *(above)*. Subacute bacterial endocarditis of the aortic leaflets which are incompetent as a result of syphilitic aortitis.

FIGURE 5 *(right)*. Embolic occlusion of both renal arteries and the aorta which occurred in a 65 year old man with mitral stenosis.

Death attributable to one or more of these cardiac causes was found to occur in 50 cases or 67.5 per cent of the entire group. Cerebral vascular disease, malignant neoplasms and bronchopneumonia, each so prevalent in the aged, are also liberally represented in Table 4. In many of this group the valvular lesion was an incidental finding.

Systemic embolism from intracardiac thrombosis was found to have contributed to death by producing infarctions of the brain, intestines and kidneys. These complications must, of course, also be attributed to the valvular lesion, particularly when there was a left atrial thrombus in patients with chronic mitral endocarditis and when there was bacterial endocarditis. An unusual embolic complication was noted in a man of 65 years who had mitral stenosis and who died of rapidly progressive renal insufficiency. Necropsy examination as illustrated in Figure 5 showed that he had bilateral renal infarction secondary to embolic occlusion of both renal arteries.

Comment

The clinical aspects of valvular heart disease in older people involve principally recognition of the deformities, knowledge of the hazards of these lesions and their sensible management. Just as in younger persons, the auscultatory findings are usually of diagnostic quality and will establish the diagnosis if valvular heart disease is suspected by the examiner. White[7] has pointed out that valvular deformity may be mistakenly diagnosed by faulty interpretation of functional apical systolic murmurs due only to cardiac dilatation and he has also noted Garvin's observations concerning the occurrence of aortic diastolic murmurs in cases of hypertensive heart disease without aortic valvular deformity. It seems likely, however, that more errors will be those of omissions in that existing valvular heart disease escapes detection or at least proper interpretation, among older patients whose unrelated presenting complaints direct

the physician's attention away from the heart. Another reason for failing to diagnose valvular disease is in the ever existing tendency to consider that cardiac disease in the older age group results from coronary disease. Sprague and Carmichael[1] have said concerning bacterial endocarditis: "Some of the difficulty in diagnosis has arisen from a lack of the recognition of the possibility of this complication in the aged; and the tendency for the picture of the subacute form to be obscured by chronic low grade infection of the lungs, kidneys and bladder." Zeman,[8] who earlier had stressed similar points in explaining the difficulty of diagnosing bacterial endocarditis in the aged, found that in one-half of his group of patients more than 60 years of age, the typical clinical picture was so obscured as to elude correct diagnosis. The hazard of bacterial endocarditis in patients with valvular heart disease is apparently a very real one for those who require prostatic resection. This complication was present in 4 of this series and has been previously reported in 8 patients at the Mayo Clinic by Merritt.[9] Recognition of this hazard should rightfully require the prophylactic use of antibiotic therapy for this type of surgery, regardless of whether or not valvular disease is suspected preoperatively.

Thromboembolic disease is another very real hazard for the older person who has valvular heart disease, especially mitral disease and particularly in the presence of cardiac arrhythmia. Under selective circumstances this danger will warrant maintaining anticoagulant therapy over a long period.

Recently 43 necropsied cases with pure calcific aortic stenosis in which the patients were more than 50 years of age were studied at the Mayo Clinic.[10] There were 18 of this group who had clinical coronary insufficiency despite the fact that severe coronary sclerosis was found in only 7. This finding emphasizes the fact that coronary insufficiency may be accentuated by aortic stenosis and may occur at times without significant coronary occlusive disease.

The threat of cardiac decompensation with congestive failure is constantly present to the older person with valvular heart disease. Congestive failure may develop insidiously simply from the valvular deformity or it may occur acutely as the consequence of the development of cardiac arrhythmia. Congestive heart failure for the older person with the valvular heart disease is managed with the usual cardiac regimen.

In spite of these pitfalls in the path of the patient with chronic valvular heart disease who exceeds 50 years of age, it must again be pointed out that a long life of such a patient is not a rarity. White and Bland[11] have reported mitral stenosis in patients of more than 80 years and our data illustrate longevity among many, particularly with aortic stenosis. In the absence of evidence of cardiac insufficiency or active rheumatism, Sprague and Carmichael[1] have pointed out that an active life has no apparent deleterious effect on the patient with rheumatic valvular damage. This last point bears re-emphasis in order that asymptomatic patients with a murmur shall not be regimented into a needlessly sheltered life and treated with cardiac pharmaceuticals and diets. It may be finally pointed out that although many old persons with valvular heart disease die of the heart disease, there are also many who live only to fade away gradually.

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Detachment of the Retina

THE SCHNEIDER EYE FOUNDATION PRESENTATION

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The etiology, pathology and therapy of detachment of the retina are outlined. Attention is called to the frequency (about 50 per cent) with which the fellow eye sooner or later becomes involved. The prodromal symptoms and the need for prompt treatment are stressed. The futility of medical therapy is emphasized and encouraging results from surgical treatment are reported.

It is in the past 20 years that detachment of the retina has taken its rightful place in the foreground of ophthalmologic thought. The beginning of this era was marked by Gonin's epochal discovery (1925-1928) that by closing the tear in the retina many of these separations could be cured. His service to ophthalmology lies not only in the therapeutic revolution he inaugurated but also in the stimulus which his work gave to investigations on all phases of this disorder. The etiology, pathology and prognosis as well as the treatment have been intensively studied and out of these researches new ideas and great hopes have emerged. It is the purpose of this presentation to familiarize you with the present day concept of this condition.

Etiology

Detachment of the retina is not a pathologic entity but is an anatomic accident which may be the result of many causes. In this condition a cleavage takes place between the two primitive retinal layers—the outer pigment epithelial layer remains attached but the inner layers become separated. For clinical purposes detachments can be classified as solid, inflammatory or serous. The solid detachments are those produced by intraocular neoplasms; the inflammatory ones are caused by a collection of exudative fluid beneath the retina; the serous type is the result of a break in the continuity of the retina which allows the degenerated vitreous body to pass underneath it and float it forward. This latter group is most common and is the one with which this presentation deals.

There are many predisposing factors in the production of serous detachment, such as race, sex, age, myopia and trauma, all of which are important to the ophthalmologist but are of only passing interest to the general physician. Suffice it to say that about two-thirds of all such detachments occur in myopes and that trauma plays some part in the production of approximately 20 per cent of them. More important is the knowledge that this is essentially a bilateral disease. If one excludes the cases resulting from frank trauma (e.g., penetrating intraocular foreign body) the second eye becomes involved in about 50 per cent of all other detachments.

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

The time interval between the detachment in the first eye and that in the second may be as long as 10 or more years but the high incidence of its occurrence is not generally appreciated. In my opinion this is the strongest reason for salvaging all possible vision in every case of detachment. It is neither necessary nor desirable to use this argument to convince the patient of the need for surgery but this knowledge should make one think twice before adopting a "laissez-faire" attitude.

Pathology

The pathologic processes underlying a serous separation of the retina are varied but chief among them are (1) cystic degeneration of the retina in the periphery and (2) changes in the vitreous body. The periphery of the retina is the seat of degenerative changes in many individuals and in some the resultant thinning is so great that breaks in its continuity take place. The vitreous body often shrinks and becomes detached from the retina. If, when this occurs, the vitreous structure is adherent to the retina at any point the traction exerted by its contracture may be sufficient to produce a tear in the retina. Either one or both of these pathologic processes are present in most serous detachments and constitute the productive mechanism responsible for the vast majority of these cases. An insignificant trauma in the presence of these pathologic changes may be the precipitating cause, but often the detachment develops spontaneously.

These pathologic processes occur more frequently in the myopic eye and with regularity as a senile change and tend to be progressive. Their progressive nature accounts for the disheartening recurrences occasionally encountered and for the frequent involvement of both eyes. Familiarity with these degenerative changes is therefore of prognostic value.

Symptomatology

The prodromal symptoms which should interest the general physician are (1) black spots and (2) flashes of light. While neither of these complaints is pathognomonic of detachment of the retina the sudden appearance of a shower of black floaters or a persistent flashing of light warrants thorough investigation. Detachment of the retina is sometimes ushered in by a hemorrhage into the vitreous which causes a sudden reduction in the visual acuity, while in others the first complaint is only a defect in the field of vision. Since the majority of detachments begin in the periphery and progress toward the central area it is important to initiate treatment promptly because involvement of the macula materially lessens the chances for restoration of good visual acuity.

Treatment

The first step in the treatment is the determination of the exact nature of the detachment. Some separations of the retina are produced by neoplasms. These are either primary or metastatic lesions. The most common primary one is melanoma of the choroid and for this, removal of the eyeball is the only treatment. Metastatic involvement of the choroid occurs most frequently with carcinoma of the breast, but of course can arise from a primary lesion anywhere in the body. Since these tumors are usually bilateral and life expectancy is short, many feel that it is futile to attempt treatment, but often the growths can be arrested by radiation with preservation of vision.

The inflammatory type of detachment may be purely an ophthalmologic problem which calls for only local treatment of the cause, or it may result from some systemic disturbance such as toxemia of pregnancy or diabetes. In neither of these latter conditions should surgery be considered. If the separation has been produced by toxemia of pregnancy prompt treatment of the cause frequently will cure it. Detachment of the retina occurring in cases with advanced diabetic retinopathy is usually the result of organization of fibrous tissue within the vitreous and is not amenable to treatment. Similarly a detachment produced by any type of proliferative retinopathy is not suitable for surgery.

Fortunately most detachments are serous in nature and great progress has been made in the management of these cases. Thirty

years ago the treatment of serous detachment of the retina was a sad story. Prolonged bed rest, exhausting pilocarpine sweats, aspiration of the subretinal fluid and many other measures were used to little or no avail. So discouraging was the outlook that most ophthalmologists advised no therapy and only hoped that the fellow eye would not become involved. Medical measures are still futile but the majority of the patients can be cured by surgery. Although a detailed description of the operative technic is not appropriate here, it is fitting to tell you its underlying basic principle. This is permanent closure of all breaks in the retina by the creation of adhesions between the retina and its underlying choroid.

Gonin's observation 25 years ago that serous detachments result from a break in the continuity of the retina has gradually been accepted until today the vast majority of ophthalmologists recognize the etiologic significance of the hole or tear in all such cases. The finding and localization of these holes is the first step in the treatment. Since the holes are frequently tiny and peripherally situated this is often tedious and time consuming, but must be done. Proper emphasis has been placed on repeated preoperative examinations, for without a thorough knowledge of the entire fundus the operation becomes a hit or miss affair.

The method of closure of the hole or holes has undergone many changes since Gonin first used the galvanocautery. This instrument he boldly plunged through the overlying sclera and choroid in the hope of creating sufficient inflammatory reaction in the region of the previously localized hole to effect a cure. The reaction produced by this traumatizing procedure was in itself so undesirable that many modifications have been tried. Among those which received considerable attention was the Guist method of chemical cauterization. In this operation trephine openings were made through the sclera over the site of the detachment. To each spot of exposed choroid, potassium hydroxide was applied. This procedure led to a larger number of cures but produced such extensive damage to the surrounding retina and choroid that the search continued for an equally effective but less traumatizing technic. The present day method of electrocoagulation is the outcome of these investigations. In this operation the area surrounding the hole can be effectively barraged with minimal destruction to the surrounding tissues. These coagulations produce localized areas of inflammation in the choroid and adjacent retina if these two structures are in apposition. Since contact is essential to cause the retina to adhere to the choroid the subretinal fluid must be evacuated. This is done by minute perforations made with diathermic needles or pins long enough to reach the subretinal fluid. In some instances, to effect or to maintain this contact, injections of normal saline solution or air are made into the vitreous cavity. This raises the tension of the eyeball, forces the subretinal fluid out through the small openings and holds the retina in apposition to the choroid until the resultant cicatrix can form.

In certain instances there is an actual shrinkage of the retina and vitreous body as well as a stretching of the sclera. If these conditions are present the usual diathermic technic will not succeed in placing the retina in contact with the treated choroid. For these cases a segment of sclera is first resected and then diathermic applications are made. This so-called scleral resection is reserved for the more desperate situations, and although the indications for its use are not universally agreed on it does constitute an important addition to our surgical armamentarium. Many so-called hopeless eyes have been salvaged by this method of treatment and the field of its usefulness is steadily enlarging.

Results

As more and more attention has been directed toward careful preoperative examinations and meticulous surgical treatment the percentage of cures has steadily risen. Twenty-five years ago Gonin's claim of 20 to 25 per cent successes was considered almost unbelievable. Today 65 to 70 per cent of these cases are being cured as evidenced by the results obtained on the last 131 patients treated in my office. We were successful in reattaching the retina in 71 per cent of them. This list

of consecutive cases includes 39 in which, owing to the unfavorable findings, the patients were treated by scleral resection. It is particularly encouraging to note that in this latter group 59 per cent of the resections were successful. In the 92 cases on which a diathermic application was used 76 per cent were reattached.

Stimulating as these results are, much still remains to be accomplished. This can be done only by continued study of the problem by the ophthalmologists with the aid of general physicians. How can you help? First, by realizing that serous detachments do not become reattached without surgery. Second, by insisting on prompt care. The longer the retina remains detached the poorer is the outlook. This does not mean that surgical treatment must be instituted at once, for a few days of preoperative bed rest are often essential. The timing of the operation depends on many factors but the commencement of preoperative treatment should be started as soon as the diagnosis is established. If, then, the general physician is familiar with the prodromal symptoms and the necessity for prompt treatment and the ophthalmic surgeon does his part, the operative results will continue to improve.

Postgraduate Clinical NOTES

2. BATHING OR IRRIGATION OF AN EYE

Bathing of the eye is indicated when there is any irritant which should be washed away, and again, if the eye is very painful, hot bathing can be soothing and comforting. To bathe the eye properly the patient should be recumbent.

Articles required in a hospital: a towel laid upon the patient's chest, another towel encircling the head, a container with the fluid that is to be used, an undine, kidney basin, and cotton-wool. The undine is filled with the fluid, the kidney basin is pressed against the cheek on the same side as the eye to be bathed, and the patient directed to tilt the head a little towards that side. A little of the fluid is run from the undine on to the back of the hand of the person who is to do the bathing. If the fluid is too hot for the back of the hand, it is certainly too hot for an eye. The eyelids are separated, using the thumb and first finger of the left hand, and the fluid is directed into the eye from a height of about 6 inches. The patient is told to look successively upwards, downwards and to the right and left. The fluid passes over the eye and is caught in the kidney basin. The lids are then wiped dry with cotton-wool.

In the home, such utensils as undines or kidney basins are not usually available, so something else has to be substituted. For the undine, a cream jug or old teapot is quite suitable, and instead of the kidney basin a small pudding dish can be used. In an emergency, balls of cotton-wool can be dipped into the hot lotion and the fluid squeezed into the eye.

J. R. Mutch, Drugs Used in Ophthalmology, *The Practitioner*, September 1952, p. 266

Problems in Thoracic Surgery

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The problems encountered in the treatment of thoracic diseases have increased as our knowledge of the various pathologic conditions has accumulated. Surgical treatment is now of a definitive nature, the attack direct and of sufficient intensity to remove the offending lesion. At our present state of knowledge any other type of therapy is mentioned only to be condemned. Constant vigilance for newer and more effective methods of treatment is obligatory if progress is to continue.

J. W. GALE

THE thoracic cage, which constitutes more than 15 per cent of the body area, was one of the last regions to be explored by the surgeon. The thorax contains two of the most vital structures in the body: the heart and lungs. Life cannot be long maintained without adequate function of these organs. Any condition which tends to upset the physiologic balance of this cardiorespiratory mechanism is most serious. Why then has this region resisted surgery for so long? There are several reasons: The normal physiology was poorly understood; adequate anesthesia to provide proper ventilation during an operation in the pleural space was unsolved, and good surgical technic was undeveloped.

The first great impetus to thoracic surgery came during World War I. It was at this time that many fundamental problems in the physiology of the chest were discovered by the commission appointed to study the treatment of empyema. Since then improvements in anesthesia and surgical technic have developed to a point where exploration of the thoracic cavity carries a mortality rate no higher than that for exploratory laparotomy. Other allied fields of endeavor have assisted in a better understanding of the problems: better methods and interpretation of the roentgenogram taken from various angles, enhanced at times by the use of bronchography, with the bronchoscope affording direct visualization of the tracheobronchial tree; cytologic studies of the cells appearing in the bronchial secretions, and, last but not least, the discovery of the antibiotics.

Many diseases affect the thorax and its contents. It is not within the scope of this paper to discuss all of them; rather, the objective is to consider their general importance to the busy general surgeon. It immediately becomes obvious that a specialist in thoracic surgery is not always within calling distance, particularly when an emergency occurs. However, if certain diagnostic and therapeutic measures are employed, there will result a great saving of life in both acute and chronic chest conditions.

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

In this age of speed and more speed, with thousands of planes in the air and millions of cars on the highways, there probably is not a physician in the United States who does not see at least one chest injury during the year. These injuries vary from a simple painful rib fracture to multiple complicated fractures, flail chest, injury to blood vessels with resultant hemorrhage and even laceration of the underlying lung or lungs. Even the heart itself may be severely traumatized.

Regardless of the type of injury, respiration is disturbed. A simple rib fracture without displacement is very painful during respiratory movements. More severe injuries call for an immediate evaluation of the patient's condition. An early correct diagnosis and adequate therapy are mandatory if the patient's life is not to be jeopardized. If shock is present, the usual measures must be taken to correct it. Properly planned roentgenologic study is invaluable in determining the condition of the underlying lung or lungs. The physical examination is most important, as is the reading of the blood pressure, but roentgenographic examination can be done while the patient is undergoing treatment for shock, and as many views as possible should be made to determine the presence or absence of blood or air in the pleural cavity. The degree of collapse of the lung can be determined and any shift of the mediastinum causing collapse of the opposite lung recognized. If this condition exists, thoracentesis is in order and should be carried out immediately to relieve the tension of hemopneumothorax, restore the displaced organs to their normal position and aid the immediate reexpansion of the lung.

Too often thoracentesis is delayed because there is fear of damaging the underlying structures. This fear is unfounded if care is taken to stay close to the top of the seventh or eighth rib in the posterior axillary line. A simple apparatus for this procedure and one which will not permit air to enter the pleural cavity can easily be kept ready at all times (Figure 1). If blood and air are obtained in quantity, simple aspiration may not suffice. A small rubber catheter should be inserted

FIGURE 1. Materials for aspiration of fluid from pleural sac: 20 cc. syringe, artery forceps, soft rubber tube and No. 18 needle.

under local anesthesia and constant suction drainage at –3 to –4 cm. of water maintained. This will encourage rapid inflation of the lung and obliteration of the dead space, and frequently stay or decrease the bleeding from the lung or intercostal vessel. Blood should be given to replace what has been lost, and the quantity can be estimated by the amount being removed by aspiration and suction. Bleeding from a lacerated lung usually subsides quickly. If bleeding continues and control of shock cannot be maintained, thoracotomy with control of the bleeding is indicated. Many patients will not require surgery if the conservative measures mentioned are faithfully carried out (Figure 2).

Pain is always present if ribs are fractured. The chest should not be strapped. The intercostal nerves should be blocked with procaine proximal to the fracture or fractures. Breathing will then be painless and not limited by restraining, uncomfortable adhesive plaster.

At times injuries will be so severe that the abdominal structures will also be involved (Figure 3). However, one must not forget the primary injury to the respiratory appara-

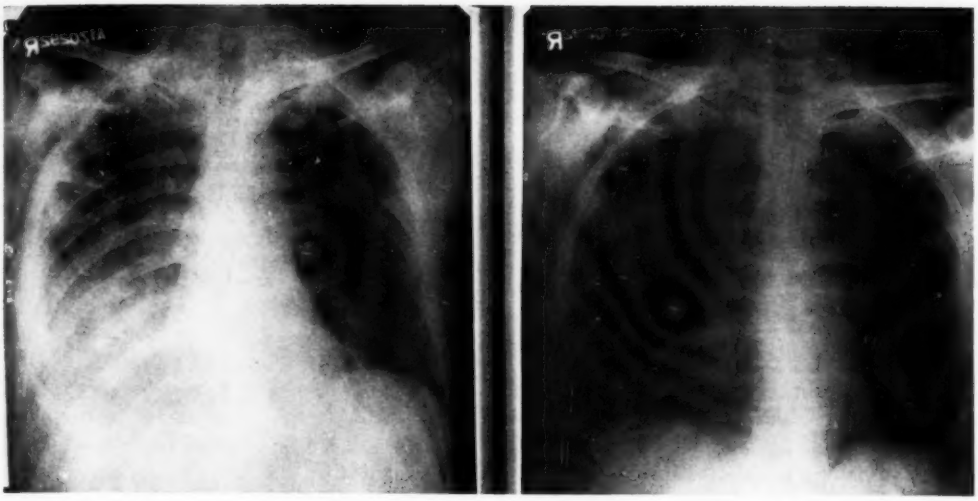


FIGURE 2a *(left)*. Roentgenogram of chest of patient injured in automobile accident. Severe shock was present. Hemothorax on right. b *(right)*. Same patient, 48 hours later. Lung completely and permanently expanded after several aspirations of blood from pleural cavity. Recovery.

tus which must be kept functioning at the highest degree possible at all times. These serious, complicated injuries draw heavily on the diagnostic and therapeutic acumen of the most experienced surgeon.

Other thoracic problems merit attention. Congenital cystic disease of the lung is relatively common and we have learned to recognize it. Cystic disease and congenital diaphragmatic hernia are frequent causes of respiratory embarrassment in newborn infants. At birth the cysts may be numerous or they may occur singly and be of large size. Once respiration has been established automatically in the infant, if air enters the cysts but cannot escape they grow larger and be-

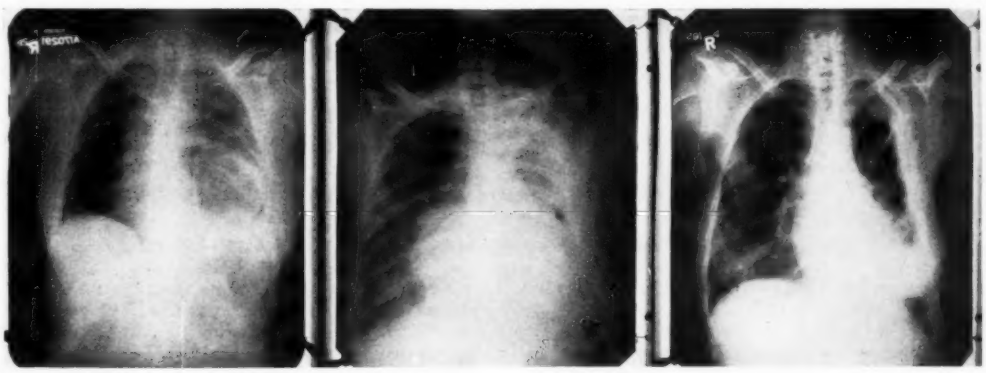


FIGURE 3a *(left)*. Multiple rib fractures on left and large gastric bubble due to ruptured diaphragm. Patient injured in automobile accident. Heart contusion and fractured spleen also present. b *(center)*. After splenectomy and repair of perforation of stomach and of traumatic diaphragmatic hernia. c *(right)*. Six weeks later, marked improvement of left lung field. Asymmetry of left hemithorax due to multiple rib fractures. Recovery.

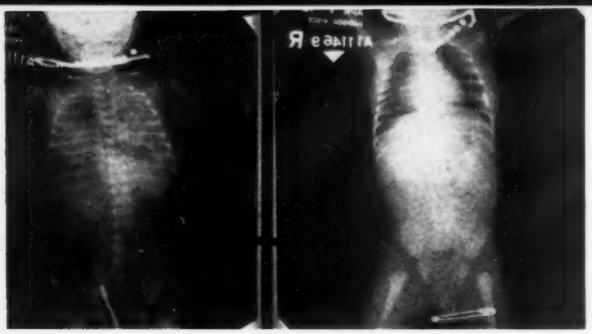


FIGURE 4a *(left)*. Congenital diaphragmatic hernia accompanied by dyspnea and cyanosis in newborn infant. b *(right)*. Postoperatively, abdominal viscera restored to peritoneal cavity; congenital defect repaired. Recovery.

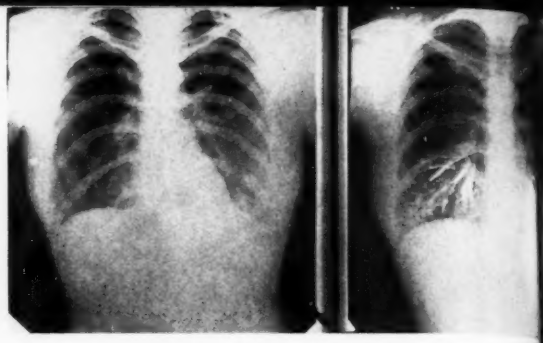


FIGURE 5a *(left)*. Roentgenogram, young adult. Little evidence of disease. b *(right)*. After injection of LIPIODOL®; extensive bilateral bronchiectasis of lower lobes. Recovery followed bilateral resection of lower lobes.

come tense, causing compression of the lung and mediastinum to a degree where functioning lung tissue is so reduced that adequate respiration is impossible. A roentgenogram affords immediate diagnosis, and decompression of the offending cyst by thoracentesis will suffice to improve respiration until permanent decompression or removal can be carried out.

Congenital diaphragmatic hernia (Figure 4) may produce similar symptoms when most of the viscera lie in the pleural cavity. The defect should be repaired at once if there is serious interference with respiration. If the defect is not producing acute symptoms, repair may be delayed but is advisable during the first year of life because of the danger of such complications as volvulus or intestinal obstruction.

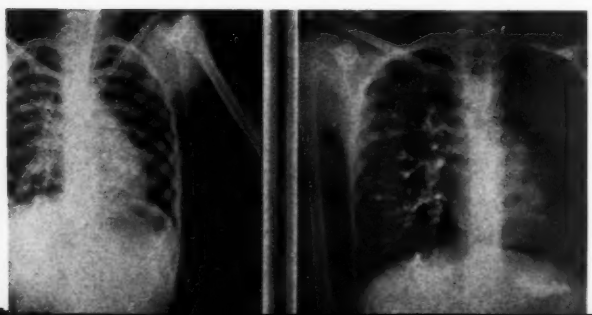
The inflammatory diseases of the lung which have attracted increasing attention are bronchiectasis and lung abscess. Their diagnosis is not particularly difficult, but the number of therapeutic measures which have been instituted have been exceeded only by the remedies recommended for a bad cold. Bronchiectasis, being a chronic disease of the bronchi and contiguous parenchyma, frequently can be traced back to its inception following one of the exanthems or pneumonia in childhood. Many persons with this condition have a copious, putrid sputum which may force them to withdraw from society. In about 70 per cent of the cases, hemoptysis occurs during the course of the disease. The presence of bronchiectasis can easily be determined through bronchography (Figures 5 and 6). It may be unilateral or bilateral. The only satisfactory treatment is extirpation of the diseased tissue (lobectomy or pneumonectomy).

Lung abscess (Figure 7), long considered a most serious and frequently fatal disease, has been attenuated by the antibiotics but is still a formidable malady. Formerly, in those cases in which spontaneous healing did not occur (50 per cent), the abscesses were drained. Recurrence and mortality rates were high. It is now well established that excision within a few months is followed by low rates of mortality and morbidity.

Mediastinal tumors present a variety of sur-

FIGURE 6a *(left)*. Lipiodol filling, right lung. No evidence of disease in 1945. b *(right)*. Five years later, injection of lipiodol reveals advanced bronchiectasis throughout right lung. Evidence of progression.

FIGURE 7a *(left)*. Chest of 39 year old man two weeks after extraction of 14 teeth under ether anesthesia. b *(right)*. Lipiodol injection shows distortion of bronchi in right lower lobe. Recovery after right lower lobectomy.



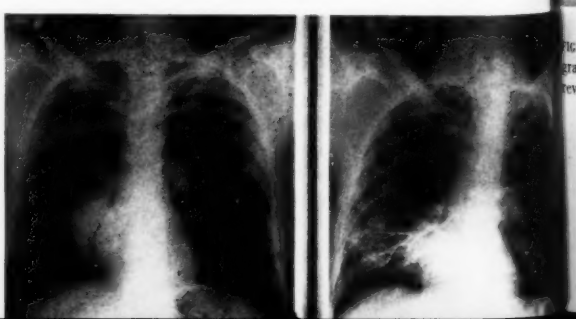
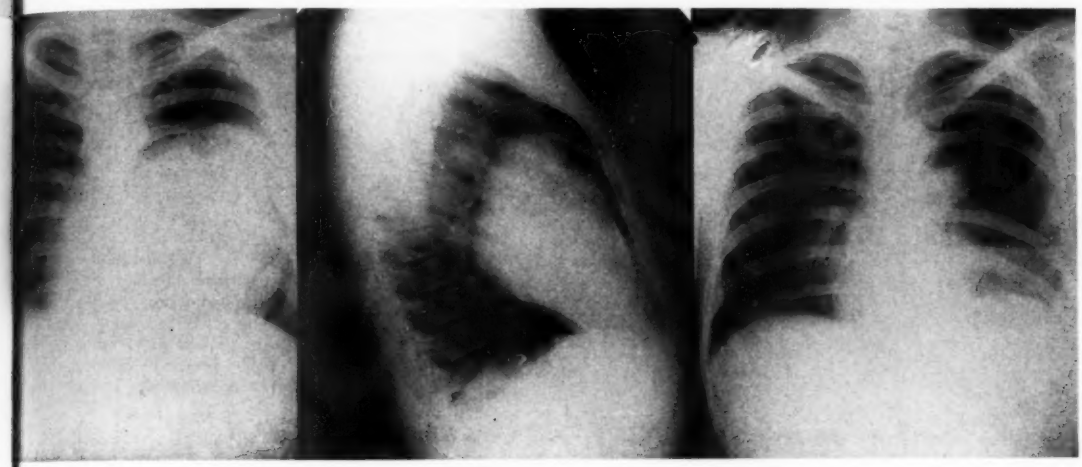


FIGURE 8a *(left)*. Anteroposterior roentgenogram of chest of 27 year old man. Complaint: shortness of breath on exertion, gradually increasing in severity. b *(center)*. Lateral view showing anterior location of tumor containing calcium peel typical of dermoid cyst. c *(right)*. Postoperative roentgenogram following removal of tumor. Fluid persists at base. Later this fluid disappeared and lung remained completely expanded.



gical problems. The most common types are dermoid cyst (Figure 8), teratoma (Figure 9), neurofibroma (Figure 10), spring water cyst (Figure 11), cystic lung (Figure 12) and spontaneous pneumothorax (Figure 13). All of these have their diagnostic characteristics on roentgenologic examination. They may be silent for long periods but the symptoms are dependent on location as well as size. Their removal is advocated because of the possibility of malignancy in the later stages. Certainly the burden of responsibility rests on the surgeon who practices watchful waiting.

IGURE 9. Anteroposterior roentgenogram, 14 year old boy. Exploration revealed large mediastinal teratoma.

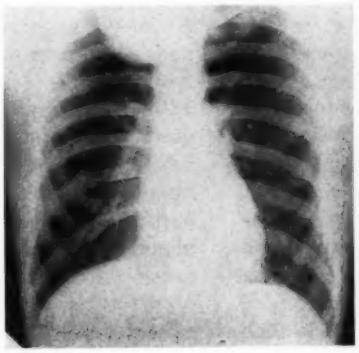


FIGURE 10a *(left)*. Anteroposterior roentgenogram of chest of 50 year old man. Large, clearly defined apical tumor easily seen. b *(right)*. Oblique spot film discloses tumor of posterior mediastinum. Tumor proved to be neurofibroma.

FIGURE 11a *(left)*. Anteroposterior roentgenogram of chest of young woman. Rounded tumor in region of aortic knob. b *(right)*. Oblique film showing posterior location. Tumor was removed; it was a spring water cyst.

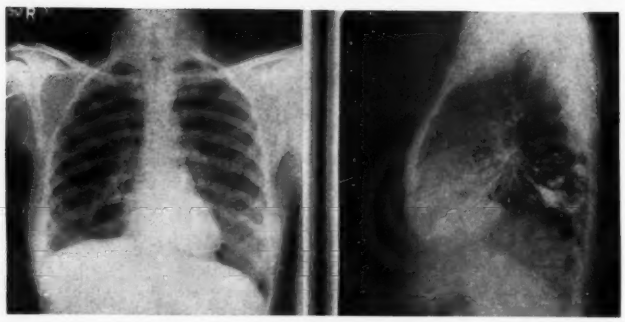


FIGURE 12a *(left)*. Anteroposterior roentgenogram of chest of 25 year old woman. Large cysts in lower right lobe. b *(right)*. Lipiodol filling, lateral view, outlining cysts in posterior basal segment of lower right lobe.

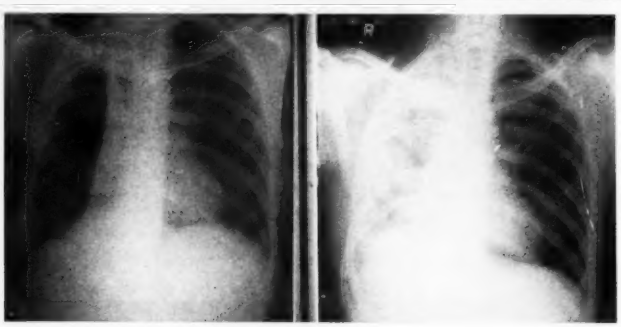


FIGURE 13a *(left)*. Anteroposterior roentgenogram of chest of 20 year old man following spontaneous pneumothorax on the right, infected. Drained. b *(right)*. Immediately after decortication. Patient gained 40 pounds in two months following expansion of lung.

Various antituberculosis associations throughout the country are providing free roentgenographic study to our citizenry in an effort to discover early tuberculous disease. If any pulmonary disease is suspected, the individual is advised to see his family physician for further study. Too often this advice is ignored. The individual feels well and finds no reason for a doctor, let alone a doctor's bill. It is not always the patient's neglect. I know of too many instances where the physician advised his patient to ignore the findings. Usually patients are asked to return in 6 to 12 months for a check-up. This is not so important in chronic disease of a noninfectious nature, but it is vital when the patient or his physician ignores a lesion which is carcinoma in its incipiency. This fact assumes increasing importance when it is realized that the incidence of carcinoma of the lung has increased over 100 per cent in the past 30 years. At present less than one-third of the patients with bronchogenic carcinoma coming to surgery have resectable lesions. Eighty per cent are resectable in the early stage before symptoms appear.

Bleeding During Pregnancy

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The number of deaths from puerperal hemorrhage has not decreased materially, despite the lowered over-all maternal mortality rate which has followed the advent of antibiotics and adequate prenatal care. Bleeding during pregnancy is discussed from the clinical standpoint, and management of the various types is outlined.

W. A. SCOTT

It may be thought that my subject is hackneyed, but I believe it is still worthy of consideration. With the advent of antibiotics the ravages of puerperal sepsis have wellnigh disappeared, and adequate prenatal care has reduced the number of deaths due to puerperal toxemia. These two developments have been the most important factors in bringing about the greatly reduced maternal mortality rates in the United States and Canada. On the other hand, the number of deaths from puerperal hemorrhage has not been decreased materially, and such hemorrhage is now a more important cause of maternal death than either toxemia or sepsis.

I have used the word "bleeding" rather than "hemorrhage" because the latter term usually implies profuse, rather than slight, blood loss. Slight bleeding, however, frequently is a warning of more serious hemorrhage that may follow, and too frequently, especially in the last trimester of pregnancy, it is not given the attention it warrants.

I propose to discuss the subject from the standpoint of the clinician. The opinions I shall express are those I have arrived at from trying to evaluate my own experience, tempered by the published opinions of others. In several instances my present views differ from those that I taught, and even published, in years past.

Bleeding during pregnancy may be incidental to the pregnancy or may be the result of it. When a woman of child-bearing age presents herself with the symptom of abnormal bleeding, the first question that should arise in the mind of the clinician is whether she is pregnant. This would appear to be a truism, but an existing pregnancy is often unsuspected by the patient, and the history may lead the clinician into the same error. This is true entirely apart from cases of falsified histories where the patient does suspect the true state of affairs. I still am amazed at how many otherwise intelligent women are ignorant of their own menstrual cycles and how frequently they are unable to give an accurate menstrual history. This is particularly true when the patient thinks there is no possibility of pregnancy. Moreover, the majority of women during their menstrual life—and, indeed, even after its cessation—interpret all vaginal

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

bleeding as some form of menstruation. In addition, when one recollects the considerable proportion of women who menstruate irregularly it is obvious that we must always be alert to the possibility of pregnancy where there is abnormal bleeding.

Prior to the advent of biologic tests for pregnancy the difficulties of diagnosis were greater than today; nevertheless, such biologic tests are not infallible. A fallacious result is sometimes obtained whether rabbit, mouse or frog is used, because of an unusual physiologic reaction of the test animal. In addition, the exact amount of time that must elapse from fertilization of the ovum until a sufficient amount of estrogenic hormone is present in the urine to give a positive test has not been determined, and the length of time that the test remains positive after the death of the ovum is not constant. Conditions other than pregnancy occasionally affect the test, and in some abnormal conditions such as hydatid mole and chorio-epithelioma, instead of the test becoming negative with the death of the ovum, it becomes positive in high dilutions. There is still place, therefore, for the clinical diagnosis of pregnancy, and if the clinician will keep the possibility in mind, mistakes in treatment will seldom occur.

Bleeding Incidental to Pregnancy

When the diagnosis of pregnancy has been established in a case of abnormal bleeding, the next question is whether the bleeding is incidental to the pregnancy or the result of it. Three occasional causes of incidental bleeding are polyps, fibroids and cancer.

A cervical polyp which previously has given rise to no symptoms may cause slight bleeding due to the hypertrophy and congestion that accompany pregnancy. When a patient is known to be pregnant and such slight bleeding arises, a threatened abortion is the most likely diagnosis. There is a reluctance in such cases to do a pelvic examination, because if the patient does abort, the examination may be blamed for it. Such patients may therefore be kept in bed unnecessarily because of a presumptive but erroneous diagnosis. Even a digital examination may not disclose a polyp if it is soft and small, and it will be revealed only by visual inspection of the cervix with a speculum. If slight bleeding persists in a pregnant patient for more than a few days such examination, I believe, is then warranted and should cause no harm if properly performed. When a bleeding polyp is discovered it can usually be twisted off without danger of causing an abortion.

Fibroids only occasionally cause bleeding during pregnancy, and when they do they are nearly always of the submucous variety. In such cases great difficulty in diagnosis arises. If the tumor that is causing the bleeding is large or accompanied by other fibroids that are intramural and do not grossly distort the uterus, the patient may be thought to be further advanced in her pregnancy than she really is. Moreover, I know of no way of determining whether the bleeding is due to a tumor in the presence of an undisturbed pregnancy or is an indication of a threatened abortion due to a fibroid. Only time can provide the answer, but conservatism is the keynote of the treatment of fibroids in pregnancy, for even when there has been prolonged or frequent slight bleeding from a fibroid uterus, many cases proceed to term.

Cancer of the cervix is a rare cause of bleeding during pregnancy, and if it is early its detection may be difficult. A cervical erosion under the influence of pregnancy often has the gross appearance of an early malignancy, and cervical smears may be difficult to interpret due to the cellular hyperplasia that is physiologic. In cases of doubt a biopsy is indicated.

The treatment of this serious complication depends on many factors: the extent of the growth, the stage of the pregnancy, the predilection of the surgeon for radiologic or surgical treatment, and the facilities at hand. A Wertheim operation is more easily performed in a pregnant than in a nonpregnant patient, but it remains a procedure for a skilled and experienced surgeon and never for the casual operator. The renewed popularity of radical surgery for cervical cancer will result in many unnecessary deaths unless the operating is done by specially trained surgeons.

Bleeding Due to Pregnancy

Let us now consider bleeding that is due to the pregnancy and not incidental to it. By far the commonest cause of such bleeding is abortion—early or late. Dogmatic rules cannot be laid down for the treatment of an early threatened abortion. As long as there is a possibility that the condition will subside and the pregnancy proceed to term, interference should not be considered. Profuse bleeding usually requires termination of the pregnancy, yet the amount of bleeding is not always an indication that the abortion is inevitable. I recently confined a patient who, at the end of two months, had such profuse bleeding with the passage of large clots and with crampy pains that on admission to a hospital a diagnosis of complete abortion was made, and the products of conception were thought to have been passed in one of the clots before admission. There was, however, no cervical dilatation, and the bleeding had ceased by the time she entered the hospital, so that no curettage was done. Such cases, however, are unusual, and when there is profuse bleeding accompanied by pain, and particularly when there is dilatation of the cervix, the abortion is usually inevitable. The difficulty of distinguishing between a complete and an incomplete abortion is so great that I believe nearly all these patients should be curetted, especially when it is now so difficult to obtain a hospital bed for readmission of the patient if it is subsequently necessary.

A more difficult problem is the patient who continues to bleed slightly for a prolonged period in spite of treatment. As already noted, a biologic test for pregnancy may remain positive for some time after the death of the ovum. It is wise, however, in such cases, not to interfere until the test is negative unless other evidence of a dead ovum or an abnormal pregnancy develops. When the ovum is dead the bleeding tends to become dark or brown in color and sometimes fetid in character. In such cases it is economically unsound to keep a patient in bed, and especially in a hospital. Where prolonged slight bleeding persists, the possible presence of a hydatid mole should be kept in mind. The uterus is then frequently larger than the period of gestation would warrant and the biologic test for pregnancy may be positive in high dilutions, although both these findings may be absent. In the absence of the passage of vesicles the diagnosis of hydatid mole still remains one of great difficulty.

In the treatment of threatened abortion absolute bed rest is the great therapeutic instrument. To this may be added sedatives for nervousness or for the relief of mild pain. Today, however, most patients believe that some sort of hypodermic medication is indicated, and if you do not give them what they call "hormones" you are thought to be either derelict in your duty or not up to date in your knowledge. I am yet to be convinced that the endocrine treatment of threatened abortion is of any value. We know that a large proportion of aborted ova show developmental defects, probably the result of faulty implantation. In the early days of endocrine therapy the estrogens were supposed to stimulate uterine contractions and progesterone to inhibit them, and the treatment of threatened abortion was large doses of progesterone. Today that practice is entirely reversed, and patients are treated with large doses of stilbestrol, or stilbestrol combined with progesterone. When I was in charge of the wards we treated a sufficient number of patients by both methods to satisfy me, at least, that neither has any influence in cases of threatened abortion.

In private practice, however, one must consider what the patients have been led to expect—and, unfortunately, this usually has been the result of exaggerated articles in lay journals. Personally I do not use endocrines in the treatment of patients with threatened abortion, and thereby probably come in for a certain amount of criticism from disappointed patients. I think it should be obvious that the use of endocrines for threatened miscarriage is futile in the latter part of pregnancy at least. If there is no vitamin deficiency the use of any or all vitamins in large quantities is equally futile.

The second cause of bleeding in early

pregnancy that we should consider is that of ectopic gestation, which is usually, but not always, evidenced by some vaginal bleeding. This bleeding ordinarily is preceded by a missed menstrual period, but this fact is often obscured because the bleeding so frequently occurs about the time of the expected period. Careful questioning, however, will elicit the fact that it differed from that which occurs during a normal period, frequently being slighter in amount and darker in color.

The greatest difficulty is in differentiating an ectopic gestation from an abortion, for which it is most frequently mistaken. It may simulate any type of abortion—threatened, inevitable, incomplete or infected. Before rupture, and I use this term to include tubal abortion, the diagnosis is exceedingly difficult. When the ovum dies or has undergone tubal abortion there nearly always is separation of the uterine decidua. This usually comes away in minute pieces, but may be passed as a decidual cast, when it is easily mistaken for a uterine abortion. I have never found a biologic test for pregnancy to be of much value in the diagnosis of an ectopic gestation. If the test is positive it is indeed evidence of a pregnancy, but the latter may be intrauterine or ectopic. When negative it is evidence only of a dead ovum, but again does not tell us where that ovum may be. The clinical findings of value are pain on moving the cervix, tenderness in one fornix, and the possible palpation of a doughy mass in the pouch of Douglas.

The procedure of greatest value in diagnosis of ectopic pregnancy is a posterior colpotomy. This I prefer to do with a sharp, large-bore needle. In multiparous patients this usually can be done without anesthesia. The uterus is outlined by bimanual palpation to make sure it is not retroverted. A tenaculum is placed on the posterior lip of the cervix, and a second one is placed on the posterior vaginal wall about an inch above the first. The vaginal wall is put on the stretch between the two tenacula, and the long needle is inserted parallel to the cervix and well into the peritoneal cavity. A vacuum is then created within the syringe by withdrawing the piston a few centimeters. The needle is then slowly withdrawn. Where there is a large amount of free blood it is easily obtained, but if the needle is withdrawn slowly the presence of a very small amount of blood in the posterior fornix is easily revealed. In these cases the blood usually is dark in color and may contain minute clots. This investigation obviously should be carried out in a hospital in order that operation may be performed immediately if the diagnosis is confirmed.

Abortion and ectopic pregnancy are for the most part conditions of the first trimester of pregnancy. Placenta previa and accidental hemorrhages are usually manifested in the last trimester. Any of them, however, may first cause symptoms in the second trimester. Placenta previa occasionally is first manifested by profuse bleeding, but usually such hemorrhage has been preceded by minor degrees of bleeding over some period of time. It is the failure to evaluate properly slight bleeding in the last trimester that frequently leads to a tragic outcome. A patient a month or six weeks before term has some slight bleeding which is thought to be indicative of premature labor; hospital beds are at a premium, and she is kept at home; the bleeding ceases, only to recur to some slight extent a few days later; this process continues until there is a sudden profuse hemorrhage, and the clinician finds himself confronted with a very serious condition. In spite of crowded hospitals, even slight bleeding in the last trimester requires admission to a hospital; and even in the hospital, before any vaginal examination is carried out, operating room facilities should be available for any necessary procedure. Soft tissue roentgenography in many cases makes the diagnosis definite.

I believe that today, if the patient is in a hospital, only one of two methods should be used in the treatment of placenta previa. In parous patients, where the bleeding has not been severe and the internal os is not completely covered by the placenta, rupture of the membranes is all that may be necessary. This allows the presenting part to press on the low-lying placenta, and labor usually supervenes in a short time if it has not already started.

Where the bleeding has been profuse, where the placenta previa is complete, and in all primiparas, unless the cervix is already dilated, cesarean section should be performed. If the hemorrhage is slight and there is doubt regarding the viability of the child, it is permissible to delay active procedures until the period of viability has been reached, but this should be carried out only in a hospital and never at home. When hemorrhage has been profuse the blood loss must be replaced by transfusion.

There are still some localities where obstetrics must be practiced without the advantages of a nearby hospital. If the clinician is confronted with a profuse hemorrhage from placenta previa under these circumstances his lot is not enviable. Rapid transportation to the nearest hospital is the first requisite; while this is being done blood loss should be replaced with a transfusion of whole blood or, if this is not available, of plasma. There is no reason why plasma should not be available even in outlying districts. Vaginal packing hides the blood loss but seldom controls it; its dangers are far greater than any possible benefit.

If the patient is in labor, with a partially dilated cervix and an incomplete placenta previa, a bipolar version and the bringing down of a foot is still a procedure of value in controlling profuse hemorrhage while the patient is being transported; this means only the bringing down of a foot, however, and not the extraction of the child. It is wise to remember that the danger of bleeding from placenta previa does not always disappear with delivery, even when that delivery is by cesarean section.

Accidental hemorrhage is sometimes difficult to distinguish from placenta previa, particularly if the patient is in labor. Usually the uterus is hard and tender, but this is not always the case. It frequently occurs in women suffering from pre-eclamptic toxemia, but it also occurs apart from this condition. Moreover, toxemia may be present with placenta previa. The external bleeding may be profuse, but if the patient is not in labor and the cervix is undilated there may be even complete separation of the placenta with only slight external bleeding. In these cases the amount of shock is out of all proportion to the visible blood loss.

When the blood loss is slight and there is no shock the differentiation of the two conditions is not of great importance, for the treatment is essentially the same in each. If the patient is in labor, especially in the case of a multipara, rupture of the membranes is advisable. If the bleeding is profuse or any evidence of shock is present, transfusion should be started immediately. I do not believe that cesarean section is ever indicated in the presence of any considerable degree of shock. Such patients should be treated by transfusion and sedation, and when the shock is overcome, the patient begins labor and section is not necessary. Cesarean section, therefore, for the most part is limited to those cases of accidental hemorrhage where the bleeding is slight or moderate and the child is alive and viable. The operation is done in the interest of the child, as further separation will result in fetal death. I can conceive of cases of severe bleeding in which the resultant shock has been overcome and labor does not supervene, where cesarean section might be indicated, but such cases are rare.

Today there is seldom any necessity for hysterectomy following cesarean section for accidental hemorrhage. No matter how dark in color or hemorrhagic a uterus may appear, its circulation is quickly re-established after it has been emptied. A few minutes' observation will usually establish this fact to the operator's satisfaction.

Postpartum Hemorrhage

I do not know whether postpartum hemorrhage properly has a place in this paper, but I shall ask your indulgence for a few observations. This complication may be the result of trauma to the genital tract, or it may occur from the placental site due to failure of uterine retraction. When due to tears of the vulva or vagina its source is easily found; when due to a cervical laceration it is not always easy to visualize the bleeding point. A good light, assistants to hold speculums,

and three or four sponge forceps to bring the whole circumference of the cervix within view may be necessary. Once the bleeding point is exposed it is a simple matter to repair the laceration. Bleeding from the top of a deep laceration, however, may be difficult to distinguish from intrauterine bleeding, and it is then necessary to remove the placenta and have the uterus firmly contracted in order to find the source of the hemorrhage. When the bleeding is from the uterine cavity it may occur before the placenta has been expelled. It is then due to partial separation of the placenta, and the immediate removal of the latter is necessary. This should be accomplished by the Crede maneuver if possible; otherwise it should be removed manually. In the majority of clinics ergonovine is given immediately after the birth of the baby, and this occasionally results in contraction of the cervix and may make manual removal of the placenta difficult. A moderate degree of anesthesia usually overcomes such difficulty.

Bleeding from the uterus after removal of the placenta is due to uterine atony, but the proper conduct of the third stage of labor and the use of modern oxytocics make this a rare occurrence. In hospitals without an obstetric recovery room and with the inadequate number of nurses which is common today, the condition of the fundus may not be observed for the proper length of time; a uterus may relax and fill up with a large amount of blood with little, if any, external bleeding, and the condition may be recognized only when constitutional evidences of hemorrhage are manifest. Where such hospital conditions obtain, and they do obtain in many excellent institutions, it is the duty of the obstetrician himself to remain for a sufficient time to be assured that there is no further danger of bleeding. Surgical recovery rooms are the rule rather than the exception; obstetric recovery rooms are equally important.

Finally, may I observe that when one is confronted with postpartum shock, in the vast majority of instances this is the result of hemorrhage, revealed or otherwise.

Postgraduate Clinical NOTES

3. RENAL FUNCTION TESTS FOR THE CLINICIAN

The object of renal function tests is to detect renal insufficiency and estimate the degree of impairment of function. The tests which the clinician should employ routinely in his practice are (1) determination of specific gravity of urine; (2) estimation of blood creatinine level, and (3) phenolsulfonphthalein excretion test. In interpreting the results of these renal function tests it is important to remember that lowered blood pressure, circulatory failure, dehydration, hydronephrosis and the presence of residual bladder urine all may lower values of renal function in the absence of intrinsic disease of the kidney.

Gregory S. Slater, *American Practitioner and Digest of Treatment*, November 1952, p. 882

Surgical Removal of Foreign Bodies with the Aid of Stereoscopic Fluoroscopy

REPORT OF A CASE INVOLVING NEEDLE IN PERICARDIAL CAVITY

CARLOS SANTOS*

University of Lisbon, Portugal

A case is presented in which a portion of a needle embedded in the pericardium of an infant about 4 months old was removed by the stereoscopic fluoroscopy technic devised by the author. Some aspects of this technic are presented. It is suggested that the technic greatly reduces the traumatism necessarily accompanying other surgical procedures for the extraction of foreign bodies, and that the danger of radiodermatitis to the surgeon who uses the technic actually is minimal.

C. SANTOS

SURGICAL removal of foreign bodies with the concomitant assistance of plain fluoroscopy is a technic which I have employed for about 30 years. For about 10 years I have also used stereoscopic fluoroscopy. I have described both the technic and some of the instances in which I have used it.[1-7] In certain cases the surgical problems I faced could have been solved in no other way. Up to the present, in almost 3000 cases, I have had no operative casualties nor any major complications. Since the details of the procedure have already been published, I shall present in this paper only a case in which the technic was used, and as the case is described, the method will become apparent. Before I proceed, however, I should like to comment briefly on the alleged dangers of radiodermatitis which might, at first glance, appear to make this technic objectionable.

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Radiodermatitis

Currently, the hazards associated with radiodermatitis seem to be a matter of considerable concern in professional circles. Oddly enough, the same practitioners would not hesitate to treat dangerously infected patients during an epidemic, nor would surgeons, similarly, shrink from carrying out emergency operations in the midst of a great disaster, even at formidable risk to themselves. Yet these same practitioners might be inclined to look with disfavor on the technic of removal of foreign bodies with the aid of stereoscopic fluoroscopy, because of the possibility that they might thereby incur radiodermatitis, regardless of the fact that in certain vital instances the method might spell

the difference between success and failure.

Actually, the danger of radiodermatitis from use of this technic is much less than would be supposed. In fact, after the performance of thousands of operations with the concomitant aid of stereoscopic fluoroscopy, with the saving of many lives and the relief of much suffering and disability, the hands of the surgeon will be marked by nothing more serious than a few brown lines on the nails and, perhaps, a few slight eczematous patches or hyperkeratosis on the backs of his fingers. My own hands, for instance, after almost 30 years of intensive use of this technic, now exhibit no more than the minor evidences of exposure mentioned above.

Report of Case

The patient was a male infant from the Azores, born October 29, 1950. He had always been healthy until, on February 19, 1951, when he was nearly 4 months old, a needle in a ball of yarn penetrated the left lateral aspect of the thorax when his mother clasped him against the yarn. The mother did not understand the child's distress and tried to place him in bed to quiet him. As she put the baby down, the needle penetrated still deeper. The child contracted his left leg and cried desperately. At the same time, he bent forward and was in great distress. When he was placed on his back, he was greatly relieved. The mother soon discovered the needle, with a thread, protruding from the baby's skin. She pulled on the thread, but only a part of the needle came with it. She understood that the sharp end of the needle was embedded in the baby's body and took him to a hospital, where roentgenologic examination disclosed a fragment of needle exactly in the same situation as when I observed it later.

The roentgenologist (Dr. Duarte Magalhães Freitas, a former assistant of mine) sent me a report of the case and asked for my counsel. I replied that, from my experience in many similar cases, I believed that steel foreign bodies, although well tolerated at first, become oxidized in the course of time and sooner or later produce a local chemical irritation, which may cause extremely disagreeable disturbances. In this case, since the foreign body was situated near the heart, it was reasonable to suppose that the symptoms of chemical irritation would appear rather soon.

In this particular case, considering the risk associated with an operation on a child but a few months old, I suggested that nothing be done for about a year, if the foreign body was well tolerated.

For 24 hours after the accident the baby had moaned and showed signs of intense suffering. Afterward he had seemed to recover and to be fairly comfortable. His mother noticed, however, that he always preferred to lie on his right side. It appeared that when he was sitting he felt more comfortable if he would bend forward. The mother concluded that the foreign body was not being well tolerated, and she decided to bring the infant to Lisbon without further delay, to have the operation performed.

I saw the baby on March 26, 1951. He was a well developed, healthy child. Only the mother, who always watched him carefully, could notice the abovementioned signs of pain.

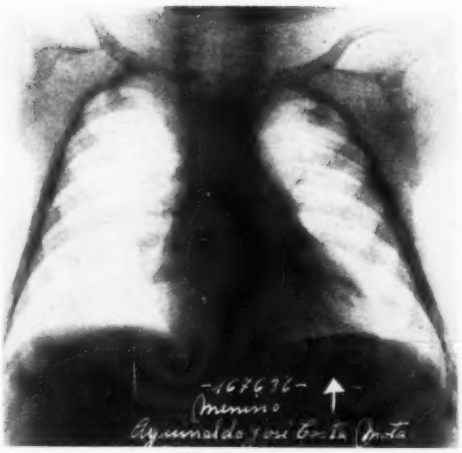
There was no clinical indication for performance of the operation at once. I considered, however, the fact that the mother was very poor and certainly would not be able to come from the Azores to Lisbon a second time. Moreover, I had evolved for this patient a special technic which would greatly reduce the danger of operation. With the mother's consent, I decided to operate.

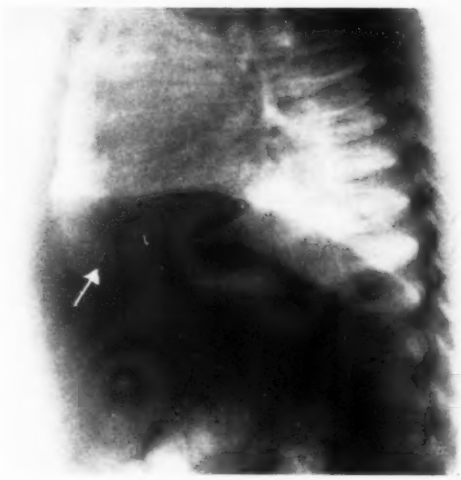
Roentgenologic examination (Figures 1 to 4) showed the needle to be situated against the inferior aspect of the heart, between the heart and the diaphragm, in the pericardial cavity, orientated from outward inwardly and slightly anteroposterior, the outer extremity being embedded in the pericardial membrane, just behind the apex of the heart. The needle followed the movements of the diaphragm and the pulsating movements of the heart. To have a more complete record of these movements, I made a kymographic study (Figure 5) with a Pleikart Stumpf kymograph, using the movable grid and fixed film. The patient was lying on his right side, and

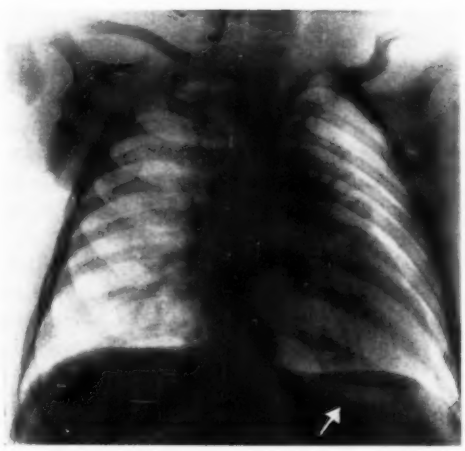
FIGURE 1 *(top)*. Anteroposterior roentgenogram made on March 26, 1951, showing the shadow of the needle superimposed on the margin of the right ventricle.

FIGURE 2 *(center)*. Lateral roentgenogram made on March 26, 1951, the needle being viewed virtually from the top, and being superimposed, as in Figure 1, on the margin of the right ventricle. The point of the needle is directed to the dorsal aspect of the patient.

FIGURE 3 *(bottom)*. In right anterior oblique projection, the needle is seen projected in its maximal length. In this view the axis of the needle is parallel to the flat surface of the film. Made on March 26, 1951.







the slits of the grid were placed horizontally—that is, parallel—to the long axis of the child's body. The kymographic image shows a larger excursion of the needle accompanying the breathing movements of the diaphragm, and a shorter and more rapid movement, corresponding to the pulsation, with two different types. At the outer extremity the movement is of ventricular type. At the inner extremity there is auricular movement.

For this kind of intervention, I use a forceps of my own design (Figure 6a), described elsewhere.[5,6] The point of this forceps opens like a crocodile's mouth. The structure of the handle ensures a firm grip. When the trigger is moved in order to open and close the point, the forceps itself does not vibrate or waver at all. Hence the movements required to introduce this forceps into the tissues can be very accurate. When operating, I make but a very small incision in the skin, just wide enough to permit introduction of the forceps into the tissues. Then, as I open and close the point of the instrument constantly, and as I press little by little, with a careful perforating movement, the forceps by means of blunt dissection opens its way through the tissues, just separating the fibers, without cutting them, making as it were but a single continuous tract until it reaches the foreign body. I make these maneuvers with the aid of the screen, using either the usual fluoroscopy or, preferably, the stereoscopic type. It is possible, therefore, to follow a straight line from the skin to the foreign body, thus reducing trauma to a minimum.

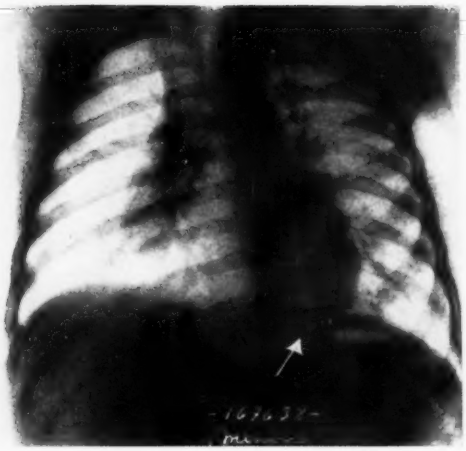


FIGURE 4 *(left)*. Roentgenogram made on March 26, 1951, in slight left anterior oblique projection, which demonstrates that the bottom of the needle coincides with the border of the cardiopericardial shadow. The absence of symptoms, plus the features disclosed in the various roentgenologic projections, suggest that the needle is situated, not in the ventricle, but in the pericardial cavity, against the lower aspect of the heart.

FIGURE 5 *(right)*. Kymographic study, made with a Pleikart Stumpf kymograph which has a movable grid, and with the slits parallel to the long axis of the patient's body. The respiratory movements of the gas bubbles in the intestines are well shown. The needle shows respiratory displacement and pulsation of the ventricular type at the outer extremity, and of the auricular type at the inner end.

In this case, there was pulmonary tissue between the skin and the pericardium. One of the possible approaches would be to pass in a straight line through the lung. As a matter of fact, I often have had to perforate the lung when removing intrapulmonary foreign bodies, and never have had an accident. Nevertheless, the fact had to be taken into consideration that, in view of the proximity of the bronchioles and the pleural cavity, deliberate perforation through a lung might initiate infection of the pleurae. In this case, since I would have to open the pericardial cavity, there was also the danger of infection of the pericardium. Under these circumstances, I thought it would be advisable to push the lung aside, by the previous performance of pneumothorax. Of course, there was the possibility that the lung was adherent to the pericardium at the site at which the bottom of the needle was embedded. In any event it was clear that every effort should be made to reduce as much as possible the possibility of contact with the lung.

If pneumothorax were established, cardiac pulsation would become more ample and the mobility of the needle would be enhanced.

A point to be noted in the extraction of a needle with my forceps is that if the needle is grasped away from its extremity the end of the needle functions as a harpoon. That is, the point will stick in the tissues and the needle will not come out. On the contrary, when the needle is grasped exactly at its extremity it becomes fixed between any two indentations of the jaws of the forceps, so that when the forceps is pulled out of the tissues the needle rotates and comes into line with the axis of the forceps. It does not become hooked in the tissues and can be extracted without difficulty.

I surmised that it would be extremely difficult, if not impossible, in this case to grasp the needle accurately at its point, because of the great mobility of the needle. I found that I could not prick the tissues in several directions until I could get hold of the extremity of the needle, for the heart was just behind it.

It was necessary, then, to find a way of reducing as much as possible the movements of the apex of the heart. For this purpose I designed a stylet indicated in Figure 6c and in Figure 7. The rear end of the stylet is bent in the shape of a ring to ensure a good hold. The anterior end is curved and its point is very sharp. It is introduced through an intercostal space to fix the pericardium by means of a maneuver indicated in Figure 7.

I found that it still would be impossible to remove the needle without the assistance of another device. The reason was that the needle was embedded in the pericardial membrane, which meant that it would have been necessary to push apart the tissues around the extremity of the needle, to permit me to be sure that I had grasped only the needle, without tearing fragments of the pericardium. The device I used for this part of the operation was a stylet (Figure 6b)[5,6] already described. This stylet was designed to be introduced through the skin so as to section the pericardium precisely about the needle and to free the end of the needle. The maneuver required for this procedure would be possible without any danger, I believed, for at this anatomic point there are no important vessels or nerves which must be avoided. Since the operation was to be performed with good visibility under stereoscopic fluoroscopy, I reasoned that I could be sure not to penetrate deeper than the pericardium at the apex of the heart, where the needle was embedded.

The operation was performed on March 30. Previously I had determined that the bleeding time (2 minutes and 15 seconds) and the coagulation time (7 minutes) did not contraindicate the operation.

The operation was performed in my roentgenologic department at the Hospital de São José, in Lisbon, where I could use my device for stereoscopic fluoroscopy.* I was pleased to have the valuable assistance of Dr. Victor Hugo de Magalhães, who administered the anesthesia, and Dr. José Maria Antunes, who performed the pneumothorax. Rectal anesthesia was produced by means of PENTOTHAL SODIUM®, followed by local anesthesia with procaine hydrochloride with epinephrine. Dr. Antunes reported on the pneumothorax as follows: "I decided that the pneumothorax had to be performed immediately before the surgical intervention, after the child had been anesthetized and was on the x-ray table.

"The needle was introduced at the axillary region. I did not notice the usual difference in sensation while passing the needle through the various layers of tissue, until the needle reached the pleura. When the needle pierced the pleura, I felt the characteristic jerk. Immediately afterward, the manometer showed well defined negative oscillations (–8; –12). I carefully introduced 120 cc. of air until I reached the pressures, +2; –2. The compression was perfectly well tolerated. The number of respiratory cycles remained practically constant. The child continued to breathe calmly.

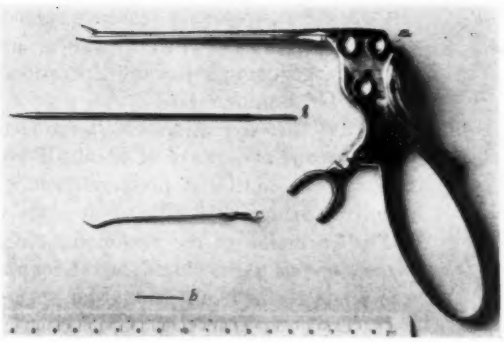


FIGURE 6a *(top)*. The author's forceps developed for the operation described herein; b. the stylet used for cutting the tissues about the needle; c. the stylet used for fixing the pericardium; d. the extracted needle.

FIGURE 7 *(bottom)*. The manner in which the stylet shown in Figure 6c fixes the pericardium.

This device is described in Santos, Carlos: Stereoscopic Fluoroscopy. In Pillmore, G. U.: Clinical Radiology. Philadelphia, F. A. Davis Company, 1947, vol. 2, pp. 671-676.

"On the fluoroscopic screen total collapse of the lung was depicted, with a gaseous cavity large enough for the operation planned by Dr. Santos.

"After the operation, I introduced the needle again and removed about 100 cc. of residual air, until the pressures again were negative (around –18)."

For extraction of the needle the child was placed on his right side. The first approach was made through an intercostal space situated very low, near the diaphragmatic contour, in order to keep well out of the lung. At the start I endeavored to determine whether or not it would be possible to grasp the needle with my forceps alone, without resort to the other devices. Use of the latter certainly would be more traumatizing than the forceps alone. As expected, it proved to be quite impossible to remove the needle with the forceps unassisted.

I then introduced my fixing stylet (Figures 6c and 7) at a point closer to the median line upon the intercostal space, placed just above the site mentioned above. While introducing the stylet, I was careful to allow only the convexity of the curve near the end of the stylet to touch the pericardium, while its point remained free in the pleural cavity. Since the diaphragm and the heart were constantly in motion, this precaution was necessary to ensure against injury to the diaphragm or the heart. Only after the convexity of the curve of the stylet had established firm contact with the pericardium did I slowly lower the stylet, until its point could touch the pericardium. Then, with a quick and careful maneuver, I transfixed the pericardium. In introducing the stylet just enough to fix the tissues, without tearing them, I was oriented simultaneously by both sight (stereoscopic fluoroscopy) and touch. From this moment on, the movement of the needle was very much reduced. The use of the stylet proved to be most efficient. At the same time, I could feel the cardiac pulsation very strongly transmitted to my left hand, which held the fixing stylet. The sharpness and force of the cardiac pulsation gave me the impression that I had perforated the cardiac muscle just at the apex. If so, this would in no way endanger the patient.

After I had well controlled the movements of the heart, I introduced the forceps through the first incision. I soon found, however, that this incision was not convenient, for it was placed too far away. I then made another opening, more internal and superior to the site of introduction of the stylet. This site was in close relationship to the costal cartilages. Tactile sensation suggested that I might have perforated one of the costal cartilages, which, at this age of this infant, are very soft. The point of perforation was sufficiently distant from the sternal border to assure that the internal mammary vessels had not been touched. After I had made the opening in the wall of the thorax, I attempted to grasp the needle, using only the forceps, without cutting the pericardium with my stylet or lancet. As I had suspected, this attempt was in vain. I then withdrew the forceps, inserted the lancet through the same opening and, relying on tactile sensation and fluoroscopy simultaneously, cut the tissues that embedded the hooked end of the needle, taking the greatest care not to cut the ventricle. When I could feel by tactile sensation that the hooked point of the needle was free, I reinserted the forceps through the same opening. Finally I was able to remove the needle without tearing any tissues (Figure 6d).

There was practically no hemorrhage. There was only a slight loss of blood from the small incision in the skin. After the operation I administered red and infrared rays with a so-called Sollux lamp, as I usually do in cases of this type.[6]

There remained the possibility that, in spite of all the care I had taken, the procedure might have damaged the ventricle; if so, creeping hemorrhage could take place during the afternoon and produce hemopericardium. To watch for such a development I placed the child in the emergency department of the Hospital de São José. At first the child seemed to feel fairly well, but some hours later emphysema developed. Following is the report of the surgeon, Dr. Fernando Frazão, who managed this complication:

"About one hour after the foregoing operation the patient was admitted to the emergency department where he was interned to detect and prevent any complication.

"The first observation shows a normal breath sound with no unusual sounds on either side. Neither cyanosis nor dyspnea was present. The child had normal sleep and normal sucking impulses. Two hours later, we noted that the scrotum was enlarged and under a certain degree of tension. There was no explanation for this development.

"About an hour later, the patient exhibited intense emphysema of the neck and thorax, and was suffering from intense dyspnea and anxiety.

"I removed the sutures from the incisions on the thorax and inserted superficially several needles in the anterior wall of the thorax.

"There was an escape of air through the incisions as well as through the needles. Simultaneously we administered oxygen through a nasal tube, at the output of 2 to 3 liters per minute.

"The condition of the patient improved gradually. He slept well and was calm for the rest of the night and the next morning."

At 10 P.M., by which time the child was already somewhat relieved as a result of the oxygen therapy, I made a roentgenogram (Figure 8) which depicted pneumothorax on the left side, subcutaneous emphysema extending to the neck and four needles inserted in the soft tissues.

The emphysema is difficult to explain. One possible explanation would be the following: It is possible that, when I had to cut the pericardium, the lung might have been adherent at that particular site, thus being damaged. If such was the case, after extraction of air performed by my colleague, Dr. Antunes, a new valvular pneumothorax may have taken place. Then since the incisions in the skin were closed, emphysema was produced. After treatment, with the wounds opened again, a great part of the air of the pneumothorax may have escaped, with the result that the emphysema was reduced. As the child coughed and cried, part of the air of the pneumothorax came out. That could explain why, when the roentgenogram was made, the pneumothorax was not very large, and no deviation of the mediastinum could be detected, which would lead one to judge that there was no intrathoracic hyperpressure. Actually, this explanation does not satisfy me entirely, but I can see no other.

Two days postoperatively the child was discharged since he showed no more symptoms.

A roentgenogram made on April 3, four days after the operation, shows that the emphysema and the pneumothorax were very much reduced (Figure 9). Another picture made on April 9 shows that all the air had been reabsorbed and that all the tissues were normal again (Figure 10).

Later, I marked with three lead pellets the spots on the skin at which the surgical approach had been made: (1) the first external incision, (2) the point at which the hook had been inserted and (3) the site at which the last incision had been made. A roentgenogram made on April 13 with the child in the left anterior oblique position (Figure 11) conveys the relationship between those sites and the corresponding intercostal spaces. Thus, it is seen that the first approach was made through the eighth intercostal space. The hook penetrated through the seventh intercostal space. The last part of the operation was performed at about the fifth intercostal space, possibly through the sixth cartilage.

A few days later a photograph was taken (Figure 12) with the child in a supine position, with the left arm raised, to check the size of the scars. The first scar is situated at about the medial axillary line. The scar produced by the introduction of the stylet is placed a little to the front, and the scar which pertains to the last incision is situated approximately on the mammillary line. The photograph gives a correct impression of the size of the scars.

I kept the child under observation in Lisbon until April 23, in order to have an electrocardiogram made, together with a phonocardiogram (Figure 13). Professor Arsenio Cordeiro kindly made this study on April 22. His report accompanied the picture.

"The general condition of the child was al-

FIGURE 8 *(top)*. Roentgenogram made a few hours after the operation, showing small pneumothorax but no deviation of the mediastinum. There is intense subcutaneous emphysema.

FIGURE 9 *(bottom)*. Roentgenogram made on April 3, 1951, four days after extraction of the needle from the pericardial cavity. Emphysema and pneumothorax are much reduced.

FIGURE 10 *(right)*. Roentgenogram made on April 9, 1951, 10 days after extraction of the needle from the pericardial cavity. The pneumothorax and emphysema have disappeared.

ways normal. In the first days after the operation he lost weight, but soon his development became quite normal. He had a good disposition and one may say that his usual condition corresponded to the expression shown in the photograph."

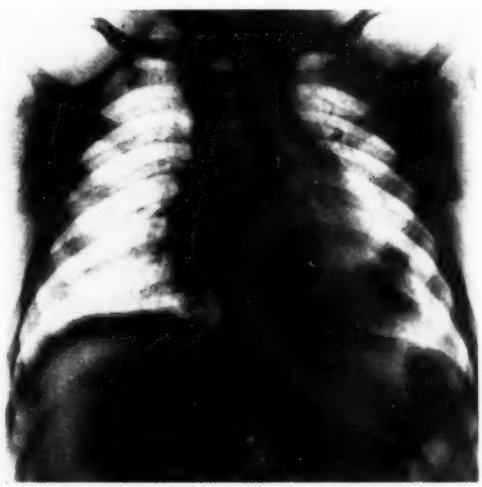
The child left for the Azores on April 23. In July and September I heard that he was in very good condition.

Comment

This case demonstrates particularly well the technic which I think should be used in such cases, a technic which I have been using for so many years.

In fact, in the foregoing case, if extraction of the needle had been performed according to the usual methods, it would have been necessary to make a large opening in the thoracic wall, to open the pericardium and, finally, to search laboriously for the needle in the pericardial cavity. In the midst of such a complicated procedure the needle very easily could be dislodged and become lost in the same cavity.

The use of radioelectric finders or detectors would not in any way reduce the magnitude of the operation itself, and would only facilitate search for the needle toward the end of



the operation. The same might be said about an operation performed with the intermittent aid of fluoroscopy as, for instance, with the Dutch device called "Boloskop," which was used by the Germans during the last war.

There is no possible comparison between the traumatism which would be produced by any of the foregoing alternative procedures and the traumatism produced by the radiosurgical technic as used in this case. Any of the usual alternative operations would be so traumatizing that most probably, in view of the relatively few symptoms in the case, no surgeon would have advised them. Thus, the child would have been doomed to suffer during his whole life from the chemical irritation produced by the foreign body. I think it is equitable to say that although theoretically it would have been possible to find a solution for this case by the usual methods, practically the problem would have remained unsolved. The radiosurgical technic allowed me to find a correct solution.

In this particular case the operation required more time than is usual for most surgical procedures carried out with the concomitant aid of stereoscopic fluoroscopy. The necessary maneuvers had to be performed very slowly and carefully, in order to prevent any damage to the diaphragm or the heart. Perfect three-dimensional visibility was a prime requisite. Another requirement was

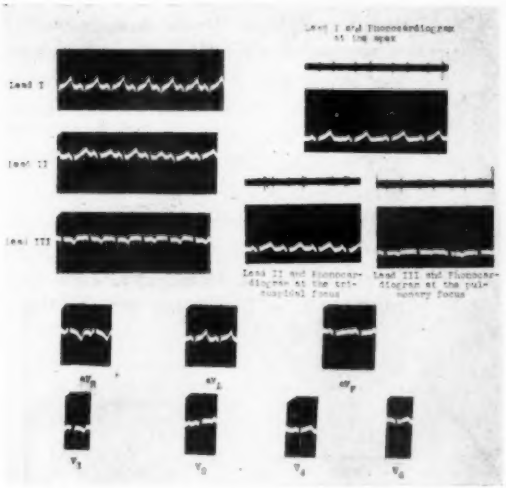


FIGURE 11 *(top, left)*. Roentgenogram made on April 13, 1951, 14 days after the operation. The shadows of three lead pellets on the skin mark the sites of surgical approach. The externally placed pellet marks the site of the first incision; the middle pellet marks the site at which the stylet used to fix the pericardium was introduced. The pellet nearest the midline of the body marks the site of the final incision.

FIGURE 12 *(top, right)*. Photograph made about one week after removal of the needle from the pericardial cavity, showing minimal size of the scars (surrounded by circles).

FIGURE 13 *(bottom)*. Electrocardiogram and phonocardiogram made on April 22, 1951, showing sinus rhythm. Electric position semihorizontal. Shape of the P waves and the ventricular complexes in all derivations is normal. There are no signs of myocardial or pericardial lesions. (Report from Professor Arsenio Cordeiro.)

protection of the surgeon's hands. The latter is achieved by virtue of the fact that the size of the beam of rays at the site of operation, as produced by my x-ray apparatus, is only 4 by 4 cm. square. This fact, in conjunction with the special shape and structure of the forceps I have described, means that the hands of the surgeon are virtually always outside the x-ray beam.

One disadvantage of the technic is that, since the field of the rays is so small, the slightest movement on the part of the patient will remove the foreign body from the field of vision. Moreover, the range in which stereoscopic vision is possible is but a few centimeters. Beyond or before that range, there is diplopia. In the case described, it was not possible to utilize deep anesthesia, with the result that the child was somewhat restless, and it was necessary from time to time to correct his position on the table. These factors combined to prolong the operation to 25 minutes, which is longer than usual.

When this technic is employed, each case must be studied very carefully, particularly in respect to anatomic considerations, which play such an important part in the surgical approach to the foreign body. Here again, however, the technic itself is of great advantage, because the route of the approach is so narrow that it is very often possible to pass between two vital anatomic structures without injuring them.

The time of surgical intervention seems to me to be of salient importance. I believe that foreign bodies such as the one in this case should be removed as soon as possible, even if they cause no symptoms. It has been my experience that if these foreign bodies are permitted to remain *in situ* for a long time, they become surrounded by the usual granuloma, which means that it will be increasingly difficult to remove them. I estimate that the delayed extraction operation is about 60 times more difficult to perform than the operation performed shortly after lodgement of the foreign body. Removal of a foreign body from the thenar eminence, for instance, usually can be done in a few seconds with the aid of stereoscopic fluoroscopy. I have done such an operation in 3 seconds. If such a foreign body were permitted to remain *in situ* for several years, on the other hand, the operation might require 10 to 15 minutes.

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PRIORITIES

In surgery, eyes first and most; fingers next and little; tongue last and least.

Sir George Murray Humphry

Acute and Chronic Radiodermatitis As Seen in the Physicist, Physician and Dentist

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Radiodermatitis is probably not so frequent today; nevertheless it is far from being nonexistent. The chronic type is seen more frequently. Occasionally a case of acute radiodermatitis is seen in the surgeon using the fluoroscope in the search for foreign bodies. The chronic cases are encountered too frequently in the internist careless in the use of leaded gloves while using the fluoroscope. Even today many dentists, especially in rural communities, continue to hold films in the mouth with their hands—and with disastrous results.

It is some 45 years since the French physicist, Becquerel, noted a peculiar reddish, burning, pruritic, localized area on the right side of his abdomen about 10 days after he had carried in his vest pocket a small plaque of crude radium, probably containing not more than 1 or 2 mg. He had carried the ore only a few weeks. Thus he discovered that radium has the ability to alter the skin with which it comes in contact.

Even before Becquerel's discovery those men who had been working with roentgen rays had learned that while these rays were a valuable agent in diagnosis, they also had some strange, unknown power to alter the character of the skin. The old-time radiologist working with a static machine had to use his own hand to gauge the proper setting of the tube and the distance from the part to be examined. Depending on how much work he did, he soon found that the skin of his hand, usually the left, became very dry and free of hair and had a sunburn-like dermatitis. It lost its elasticity and became wrinkled and, as time went on, even bound down to the underlying bony structures. There no longer was sensible or insensible perspiration in the hand, and the nails were split, broken off and atrophic.

Many years ago I had as a patient a physicist who was among the first to work with these mysterious rays. The skin of his hands, especially of his left hand, showed the changes already described, but his main complaint was loss of hair and the presence of growths in his mouth. He told me that in his enthusiasm for this new physical instrument he had been conducting a long series of experiments using his own head as the target. He wished to see if it would be possible to use this new agent for diagnostic study of the skull. Examination revealed profusely scattered, raised, whitish papillomas on the tongue and mucous membrane of the entire mouth. The affected areas were treated by means of endothermy under general anesthesia and the lesions cleared up temporarily. Many keratotic lesions on

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

the hands, the left one in particular, were treated in like manner, using local anesthesia. Shortly afterward he returned to Boston for further investigation of this new agent. He was greatly worried and no doubt he has paid the penalty for his investigations—a victim of the pursuit of science.

As early as 1896 Marcuse[1] called attention to the dangers incident to exposure to the roentgen ray—the production of alopecia and of dermatitis which resembles sunburn but is more persistent. In 1900 Kienböck[2] of Vienna gave a careful description of the changes incident to roentgen ray burn and described methods of preventing these reactions, on parts of the body near that exposed, by the use of lead. Oudin[3] in 1897 reported on the first case in a physician, and in 1907 Porter and White,[4] dermatologists in Boston, were able to collect reports of 10 cases of roentgen ray cancer in physicians, 6 of them fatal, and they added 2 of their own. One case was that of a man who began working with roentgen rays in March 1896; symptoms of chronic roentgen ray change became manifest by October of the same year, and in a period of five years, 10 epitheliomas were seen. In Porter and White's second case a spindle cell sarcoma originated from a long-standing roentgen ray ulcer of the ring finger. However, much damage already had been done by this new physical instrument, and much time was required for knowledge of the essentials of protection to become known throughout the world. In fact, in the early days of the use of the roentgen ray there was many a martyr to this branch of scientific endeavor and the result was a long list of seriously crippled investigators. Additions to this list are still being made.

Skin Changes Following Careless Exposure to Roentgen Rays or Radium

The erythema resulting from a dose of roentgen rays or radium may persist for days or weeks and be followed by deep pigmentation. The dose required to produce such a reaction is variously estimated at 375 to 400 r with superficial therapy. In fact, if the dose is too large, epithelitis or mucositis may result. This high dosage is necessary in the treatment of carcinoma of the mouth or of the cervix, but great care is essential in estimating the individual doses in order that the skin or the mucous membrane will have sufficient circulatory reparative properties to recover. With border gamma rays, changes will occur more slowly, but eventually the result will be the same if too much is given.

Ordinarily, in superficial roentgen ray therapy it is not desirable that an erythema be produced; rather, from 38 to 50 or 75 r, unfiltered, is given per treatment, this dose perhaps repeated once every two to three weeks for several times. Such exposures will do no harm to the skin and may be very beneficial in certain dermatoses. However, if such a dosage were employed over a sufficiently long period of time, the skin would show evidence of damage from radiation. Or, again, if the physician or dentist is carelessly exposed even to but a few roentgens daily or every few days over a sufficiently long period of time, his skin so exposed will in time show evidence of this damage.

In chronic cases these changes may occur very slowly and be most insidious. The first evidence may be a dryness of the skin of the exposed parts, e.g., the hands. They no longer perspire and the skin no longer has the normal amount of oil. Later the skin loses its elasticity and becomes bound down to the underlying structures, and transverse cracks are seen over the joints, particularly on the digits. Later still, there may be keratoses on the hands; these drop off only to recur and become larger and perhaps even degenerate into malignancy and ulceration. Scattered pigmentation and telangiectases are seen.

The changes in the nails of the careless user of the roentgen ray are quite characteristic. The nails become dry and hard, lose their normal sheen, are difficult to cut, and split and break off irregularly at the distal ends.

Microscopic Picture of Chronic Roentgen Ray or Radium Dermatosis

One is struck at once by the complete disappearance of the pilosebaceous apparatus

and sweat glands. These parts seem the first to suffer. Throughout the true skin there is modified or degenerated hyaline collagenous material which accounts for the density of the tissues. The elastic tissue fibers are not so affected at first. The smooth muscle tissue disappears or decreases in amount because of vacuolization and distention of the cells with hyaline change.

The greatest change in the skin is seen in the arteries and veins in chronic roentgen ray dermatitis, the intima of the arteries being greatly thickened. Wolbach states that the intima is composed of connective tissue with much collagenous intercellular substance. The elastic intima is also divided, thickened and replaced by connective tissue. The tunica media is greatly thickened and, in fact, the entire artery may be replaced by connective tissue and fibroblasts.

Wolbach states the belief that the newly formed telangiectases develop from pre-existing capillaries in chronic cases of radiodermatitis. In other words the normal elastic skin no longer expands or contracts with the movements of the joints. No perspiration occurs; the nails become dead and lifeless, and break off; the skin becomes bound down to the underlying bone; keratoses develop which often degenerate and become malignant; one digit after another may require removal. Fortunately, due to the blocking of the lymph channels and vessels, generalized malignancy is rarely seen today. In my own practice I have never seen such a case although I have seen many, many cases of chronic radiodermatitis.

How Does Radiodermatitis Occur Today in the Physician?

The question arises, how can this happen in this age of knowledge and protection? Strange as it may seem, many physicians using roentgen rays for treatment do not have a room lined with lead; they simply stand behind a leaded screen. In fact, this screen may not even extend to the floor. Moreover, the operator may be careless and not always take care to have his whole body behind the leaded screen. I know of such an example—a renowned dermatologist: He was busy and somewhat careless at times in taking full advantage of protection. Keratoses began to develop on the distal half of his right foot. Despite their removal, these keratoses recurred and malignancy developed. The deep circulation was involved and two-thirds of the foot was removed. But this was not enough. Malignancy spread higher up in the nodes and the physician died.

Again, a further type is seen—and too commonly, for many, many physicians use the fluoroscope: The doctor is rushed, has an office full of patients, just can't lay his hands on his leaded gloves and thinks to himself, "Well, this once won't make any difference." He proceeds to examine his patient with unprotected hands and wrists. True, once will not make any difference if it is a short examination. Remember, however, that this is a cumulative process, and one slip a week, a month or a few times a year gradually will build up into sclerosed vessels and further trouble. I have seen many physicians who were victims of this type of carelessness. The fluoroscope never should be used without leaded gloves. I venture to say that many physicians reading this article have keratoses on their hands, nail changes and perhaps definite vessel changes.

Another situation I wish to portray is that of the surgeon using the fluoroscope in searching for foreign bodies, e.g., a needle or a piece of steel in a patient's hand. The doctor is deeply interested in the hand; he thinks he has found the foreign body but no, he must look further; before he realizes it he has been overexposed and has suffered minimal or even severe damage to his vessels and to his skin. Just such a situation is typified by the following case: Dr. X. consulted me because of a peculiar stinging in the skin of his hands and digits. No, he had not been working in the yard with chemicals or plants, nor had he been in the woods. He had not used any new or unusual chemicals in scrubbing up. But, on further questioning, "Yes!" he recently had performed an operation on a hand during which there was a rather prolonged search for a piece of steel. Within a week he was suffering so much that he required codeine,

and the skin of the fingers of one hand showed a dark, reddish cast with epithelitis, especially the last three fingers, and in a few days one finger had to be removed. The process was very slow in its response to treatment and the pain was excruciating. He finally was successfully treated in Chicago with a radium emanation ointment.

Still another circumstance under which a radiodermatitis may occur frequently concerns both old-time physicians and dentists. I refer to the pernicious practice of holding film in the mouth when making roentgenograms of the teeth. True, such a practice is not necessary nor is it called for, yet even today radiodermatitis occasionally is caused by such action. The physician or dentist taking roentgenographic pictures of the teeth should employ the latest appliances and technics for radiographs of the teeth; they are practically foolproof.

Finally, these dermatoses from radiation are not at an end. Between the fluoroscope, radium emanation apparatus and the use of various fissionable substances there are bound to be further cases unless the greatest precautions are taken. In 1945 there were two deaths as a result of an accident with fissionable material and several of those who survived had severe reactions. I still think, however, that the fluoroscope will be the greatest factor in the production of these unfortunate but preventable reactions.

Treatment of Radiodermatitis

The war has taught us a great deal about plastic surgery, and the plastic surgeon today can do much to remedy or certainly alleviate many of the sequelae of radiodermatitis. When we encounter such a case we turn to the plastic surgeon.

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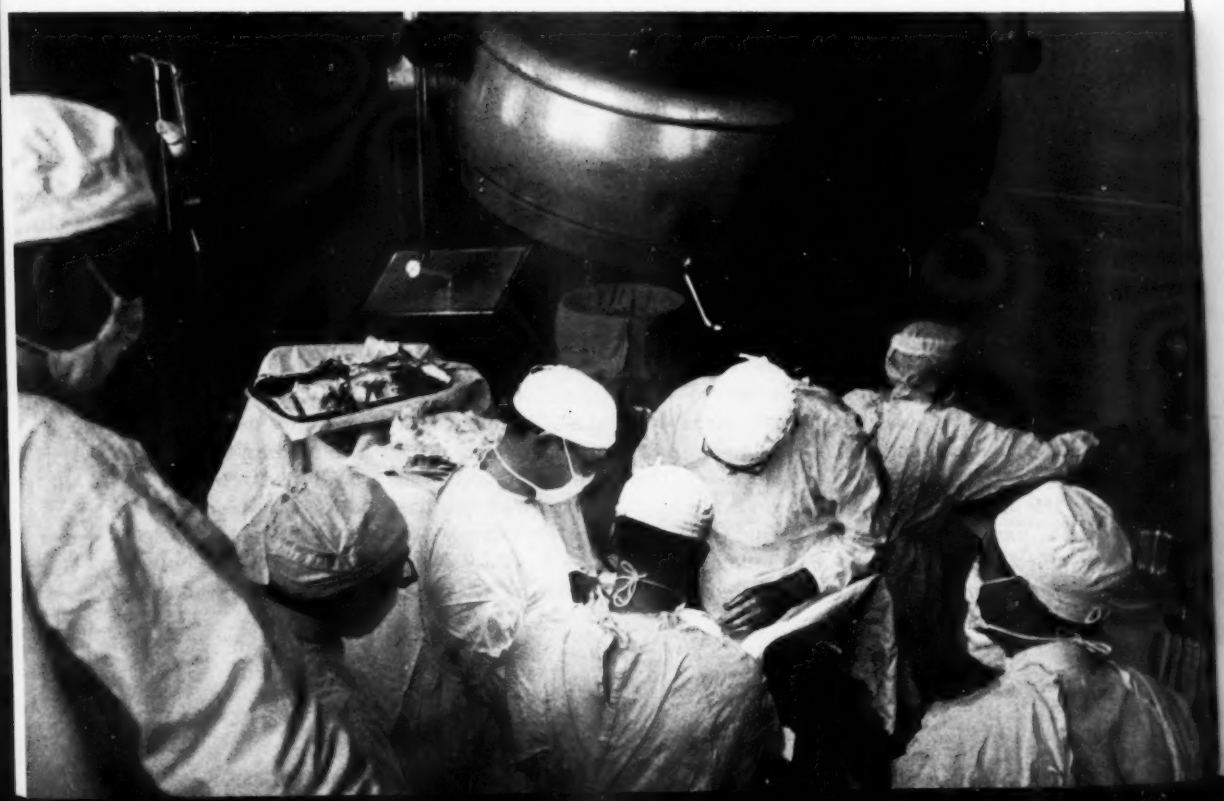
In the operating room, the results of medical research reach their highest point: the saving of human life. (Photograph by Robert M. Mottar reproduced courtesy *The Johns Hopkins Magazine*.)



Thyroid Diseases

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A general discussion is given of diseases of the thyroid from the surgeon's standpoint. Use of radioactive iodine in treatment of thyroid malignancies and of hyperthyroidism is commented on. The transition in present day hyperthyroidism from that of 25 years ago is presented. It is emphasized that 80 per cent of all thyroid enlargements are surgical lesions, and these are classified.

R. S. DINSMORE

NINE years ago a physician called me and asked that a patient of his, a man with a solitary adenoma of the thyroid who had experienced the sudden onset of stridor, be admitted to the hospital. My resident confirmed the findings of a firm adenoma of the thyroid, cyanosis and marked stridor. The patient's condition promptly improved with sedation and the use of an oxygen tent, but bronchopneumonia supervened and he was quite ill for 8 or 10 days. Examination disclosed a firm solitary adenoma and a palpable mass above the right clavicle and apparently descending beneath it, and a recurrent laryngeal paralysis of the right side. Because of this unilateral paralysis and the solitary adenoma, malignancy of the thyroid was suspected. Recurrent laryngeal paralysis from any other type of goiter, even intrathoracic or substernal, is extremely rare.

When the patient was sufficiently recovered to undergo surgery, exploration was done. A tumor was found on the posterior aspect of the thyroid; this tumor fused into the mass below. I performed a lobectomy but was unable to mobilize the tumor mass situated below, which was continuous with the right lobe of the thyroid. It was located around the great vessels and absolutely fixed. Pathologic examination established the diagnosis of adenocarcinoma of the thyroid. The tumor was studied repeatedly, and subsequent cuttings showed areas in which the tumor was almost medullary in type. Roentgen ray therapy was given, and much to my surprise the entire tumor disappeared. My experience with this type of therapy in cases of thyroid tumor as a rule has been disappointing, but nine years have now elapsed since this man received roentgen ray therapy, and although he does have a slight telangiectasis over his neck, no mass is palpable and he has no symptoms. He has carried on an active law practice and lived a normal life.

Oddly enough the late roentgenograms of his neck showed a small shadow just at the edge of the clavicle, and some nodules of calcium have appeared in this area. This finding is very significant, because calcium deposits in any thyroid tumor that is suspected of being malignant is reasonable evidence that subsequent studies will prove the tumor not to be malignant.

I am sure this man is cured; at least he

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Presented before the thirty-seventh annual Assembly of the Interstate Postgraduate Medical Association at Cleveland.

has had no symptoms and there has been an almost complete disappearance of the small shadow after nine years.

Our experience with radioactive iodine in the treatment of malignancies has been most disappointing. In only one or two instances have we felt there was any response to this form of treatment. One striking case, however, was that of a young man who had a solitary nodule in his neck which proved to be malignant. About six or eight months after its removal, the nodule recurred; again it was removed, but again it recurred. At the time of the third recurrence, the uptake of radioactive iodine was markedly increased and large marble-sized nodules appeared throughout both lung fields. In desperation we administered radioactive iodine and much to our amazement the mass in the neck completely disappeared and the nodules throughout his chest decreased in size, as proved by superimposition of the roentgenograms.

At present these nodules appear to be sclerotic. They have persisted, but the patient has been perfectly well with the exception of a slight case of hypothyroidism for five years. No new nodules have developed and those in the lungs have remained fixed in size.

I think that in malignancies as a whole you are compelled to and should use roentgen ray therapy, and the radioactive iodine uptake should be measured in all such cases because in the rare instance there will be a response to one or the other form of treatment.

One of the enigmas of thyroid malignancy as I see it—and I don't understand it at all because occasionally I am told that we simply didn't recognize these cases previously—is that in the past 10 years we have seen an astonishing number of papillary tumors in young persons and in children. Dr. George Crile, Jr. has written a great deal on this, but it is a new experience to me. We are told sometimes that possibly some of the cases that we formerly classified as aberrant thyroids actually were papillary tumors, but I refute that with the argument that we simply never saw that many aberrant thyroids—we never saw these tumors that occur in children in chains of glands up and down the neck.

I am struck by the fact that the incidence of exophthalmic goiter, Graves' disease or diffuse goiter with hyperthyroidism—whatever you wish to call it—has decreased markedly. Its severity and its incidence have changed completely! My colleagues from various parts of the country, including Dr. Donald Guthrie, Dr. Frederick Coller and the late Dr. Roscoe Graham, also have noticed this decreased incidence.

There is no question, however, that we are dealing with a mild form of hyperthyroidism at present as compared with 35 years ago, and I think we should remember this point in appraising all the thiourates and radioactive iodine in connection with definitive cures of hyperthyroidism. Formerly these patients were brought in in ambulances, were wildly delirious and had high temperatures and pulse rates, and in spite of everything you could do, as many of them died preoperatively as postoperatively. Between 1924 and 1930, 50 of our patients died preoperatively. I analyzed the reports of those 50 cases. Postmortem examination had been carried out by Dr. Allen Graham in many of them, but no important information was obtained. The report was always the same: diffuse hyperplasia of the thyroid gland, and chronic passive congestion of the kidneys, liver and spleen.

However, many patients with hyperthyroidism were carried through these crises during those years, and the most effective measures were the use of iodine painted on the skin, adequate fluids, sedation and whole blood transfusions. Many such patients were prepared for multiple operations.

At present we never see such severe cases of hyperthyroidism. My past seven residents have never witnessed a thyroid crisis. Today's patient is ambulatory when he is admitted; he may have some myasthenia, but the patient in wild delirium and with a temperature of 106 or 107° F., nausea, vomiting and diarrhea has literally passed out of the picture.

Formerly, many cases of hyperthyroidism were undiagnosed. The patient who had lost weight, was nervous and had very small glands and perhaps tachycardia was not suspected of having hyperthyroidism. That type of patient

no longer is seen. Instead we are dealing with a milder form of hyperthyroidism. True, we encounter severe cases occasionally, but the postoperative crises are practically nonexistent. Since the war I have seen only two, and both of these in consultation. Oddly enough, the same surgeon was in attendance in both cases, and both were nodular goiters and unrecognized hyperthyroidism. Both patients had undergone operation without preoperative lugolization.

During the last 10 years we have tried all the thiourates in an attempt to find a definitive cure for hyperthyroidism. Each came along with rather extravagant claims that this was the answer, that hyperthyroidism would be cured with this one. Statistics for cure started in the high nineties and gradually came down, and if you analyzed the series carefully you found that the best you could get from any thiourate was about 50 per cent in treatment of hyperthyroidism. Of course that is unsatisfactory. At least 60 per cent, perhaps nearer 70 per cent, of the cases of hyperthyroidism today include a nodular goiter, and in these cases the tumor remains.

Thiouracil itself was a very dangerous drug. It caused enlargement of the gland; in 1.5 or 2 per cent of cases, bone marrow depression resulted, and some of these patients died. Propyl thiouracil is much safer. It does not produce the agranulocytosis which accompanies the use of thiouracil. Methyl thiouracil was supposed to act faster, but actually has no advantage over propyl thiouracil. Even with propyl thiouracil in selected cases, I don't believe definitive cure can be produced in more than 50 per cent, particularly in small glands with moderate hyperthyroidism.

It has been some 10 or 11 years since Hirsch first suggested the use of radioactive iodine, and I must admit that my first impression of this agent and of the results it would produce were entirely erroneous. Initially I felt that the same clinical results could be obtained with radioactive iodine as those produced by prolonged therapy with Lugol's solution or iodine. After all, you can pretty nearly cure a case of hyperthyroidism in some instances with Lugol's solution. We used to see many patients who had been taking iodine for long periods of time and who ended up with three things: a slightly increased basal metabolic rate, but a gain in weight; an entirely satisfactory physical condition, and a lessened amount of myasthenia. But certain subjective symptoms remained: nervousness and weakness, and an uncontrolled cardiac status resulting in a persistent afebrile tachycardia. Following operation, those patients went on to a complete cure. It was my impression that patients treated with radioactive iodine eventually would present the same picture. That was not true, and I must confess that the most effective antithyroid agent I have seen used is radioactive iodine. In the first place the patient demands it, and this is one instance in which you cannot tell a patient, "You must remember that you can't be the patient and the doctor both." These patients often come to me and say, "I have come for radioactive iodine. My doctor has told me about it and I have read about it, and if it doesn't work I'll come back and let you take out my gland."

It should be pointed out that radioactive iodine should be used in exophthalmic goiter or Graves' disease or diffuse goiter with hyperthyroidism, and not in nodular goiter with hyperthyroidism, because in the latter the results are in no way comparable. If radioactive iodine is given to a patient who has a symmetric enlargement of the thyroid gland with all the symptoms of exophthalmic goiter, you can be reasonably sure that eight weeks later the result will be comparable to that following subtotal thyroidectomy. I admit that begrudgingly, but anyone who has ever done much thyroid surgery knows that leaving one-eighth of the same substance in the gland was not the answer to exophthalmic goiter. I am certain that subtotal thyroidectomy was the best method available, though I have always felt there must be some other way of treating this weird disease.

It is unfair for a surgeon to condemn the use of radioactive iodine unless he actually has analyzed some of these cases himself. I reviewed with Dr. E. P. McCullagh and some of his co-workers some 200 reports of cases

that were pure exophthalmic goiter. These 200 patients had been treated six months earlier. I found that of the 200 (and many of them were my patients), 120 had taken one drink of radioactive iodine and were clinically well; another 60 had taken two drinks. Well, if you can take something in a third of a glass of water and come up with such a result, then you must admit the agent has merit.

In that group of 200 patients there were 4 or 5 who did not respond to radioactive iodine. Paradoxically enough, the enigma was solved when the gland was removed, and each patient had a good clinical result—simply another of the riddles of this gland.

The response is quite different, however, if radioactive iodine is used to treat patients with nodular goiter and hyperthyroidism; it is slow, and often it is nonexistent. Not only do you have to give them multiple doses over long periods of time, but also the nodular goiter remains. These patients go on to cure if they are promptly operated on.

Although there are disadvantages to the use of radioactive iodine, the initial criticism that the thyroid gland would be completely destroyed or ablated and the incidence of myxedema would be high is not true at all. A very small dose—6 or 7 millicuries—does not produce hypothyroidism. In 1 per cent of cases the administration of the drug must be discontinued because of a transient hypothyroidism, and a little thyroid extract must be given, but apparently in that small dosage there is as a rule an ample amount of thyroid left to carry on normal activities, so I think the danger of myxedema is a myth.

One of the disadvantages is the specification set up by the Atomic Energy Commission, as a result of which the agent is relatively unavailable. Another criticism was based on the fact that in some experimental animals which had received huge doses, carcinoma developed; thus it was stated that cancer might develop in humans later in life. I don't know about that, but as a safeguard I do not give radioactive iodine to patients under middle age; I go ahead and operate.

Bear in mind that 80 per cent of all thyroid enlargements are surgical lesions. Year in and year out in a thyroid clinic you will find that about half of the cases are nodular goiters, adenomas or multiple colloid adenomas; another 25 per cent, and probably nearer 30 per cent, are nodular goiters with hyperthyroidism, and operation is indicated in these cases. The remaining group, 20 per cent, may be treated with radioactive iodine.

MEETINGS AND POSTGRADUATE COURSES

American College of Surgeons: Sectional meeting, February 23-24, at the Atlanta Biltmore Hotel, Atlanta, Georgia. Chairman of the Committee on Arrangements is Dr. William G. Hamm of Atlanta. An added feature is the concurrent Atlanta Postgraduate Medical Assembly; there will be three joint sessions of the Assembly and College meetings. Subjects to be covered include symposiums on trauma, gynecology, cancer and gastrointestinal bleeding.

University of Minnesota: Continuation course in clinical dietetics, March 2-4, at the Center for Continuation Study. Program will be under the direction of Miss Gertrude I. Thomas, Director of Nutrition and Professor of Dietetics.

University of Colorado School of Medicine: Course in applied medical science (spring quarter), March 23-June 8. Subject matter has been divided so that any subdivision can be taken without registering for the full course. Course is under the supervision of the Graduate Division of the School of Medicine, and the guest lecturer will be Dr. DeWitt Stetten, Jr. of New York City. Director of the course is Dr. Joseph H. Holmes.

Albany Medical College and Albany Veterans Administration Hospital, Albany, New York: A series of 18 seminars on recent advances in medicine and the basic sciences are being presented for both general practitioners and specialists on Thursday afternoons through May 7.

The Diagnosis and Treatment of Congenital Cardiovascular Defects in Infants

ROBERT F. ZIEGLER,* CONRAD R. LAM** AND RICHARD E. SHIPLEY*

Henry Ford Hospital, Detroit

An attempt has been made in this exhibit to show pictorially the most important anatomic, clinical, roentgenographic, electrocardiographic, physiologic and surgical data in the more common operable congenital cardiovascular defects in infants. Particular attention should be directed in each defect to the most important diagnostic procedures listed with the clinical features and physiologic data. It should also be noted that while the diagnostic criteria and operative procedures are given particularly for infants, they are also generally applicable to all ages. The main thesis of this exhibit, however, is that an accurate diagnosis *can* be made even in early infancy and that surgical correction *is* frequently necessary and *may* be accomplished safely and successfully in this age group.

While this is not intended to be a complete discussion of the diagnosis and treatment of congenital cardiovascular defects, certain specific points deserve emphasis, particularly from a correlative point of view. Of importance, for example, is a comparison of x-ray and electrocardiographic evidence of unilateral ventricular enlargement. In this regard, note the variations in the x-ray configuration of the heart with left ventricular hypertrophy as illustrated in patent ductus arteriosus, coarctation of the aorta and tricuspid atresia. It seems quite apparent that some other

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index of unilateral ventricular enlargement is necessary. This is to be found in the precordial lead electrocardiogram which, more accurately than the extremity leads, gives evidence at any age of right or left ventricular hypertrophy. Note too the discrepancies between the position of the mean electrical axis and precordial lead evidence of right or left ventricular enlargement.

While most of these defects, especially when "typical," may be diagnosed accurately by clinical means, physiologic studies are frequently of great importance and are as applicable in infants as in older children and adults. An outstanding example of the application of physiologic studies is in the differentiation between valvular pulmonary stenosis with an intact ventricular septum but with an auricular defect and tetralogy of Fallot. Cardiac catheterization together with a determination of the circulation time from the right ventricle to the face, as illustrated on page 151, serves to make this differentiation.

The question will inevitably arise, when *can* or when *should* operation be done in each of these various defects? While no completely satisfactory answer can be given to cover each individual problem, answers according to our point of view may be found in Table 1.

TABLE 1

DIAGNOSIS	TYPE OF OPERATION	MAY BE DONE	SHOULD BE DONE	YOUNGEST SUCCESSFUL CASE IN HENRY FORD HOSPITAL SERIES
Aortic ring	Division	Soon as diagnosed	Soon as diagnosed	3 wks.
Patent ductus arteriosus	Ligation or division	Soon as diagnosed	Soon as diagnosed	6 wks.
Coarctation	Resection	As early as in trouble	2 years plus or earlier if necessary	16 mos.
Valvular pulmonary stenosis	Valvulotomy	As early as in trouble	As early as in trouble	7 mos.
Tetralogy of Fallot	Blalock or Potts anastomosis	As early as in trouble	2 years plus or earlier if necessary	9 mos.
Tricuspid atresia	Blalock or Potts anastomosis	As early as in trouble	After 15 to 18 months if possible	5 mos.

It is obvious that all of the important details cannot be included in a study of this sort. Its main purpose is to serve as a correlated outline. For purposes of further reference a selected bibliography has been added.

LESIONS NOT ASSOCIATED WITH CYANOSIS

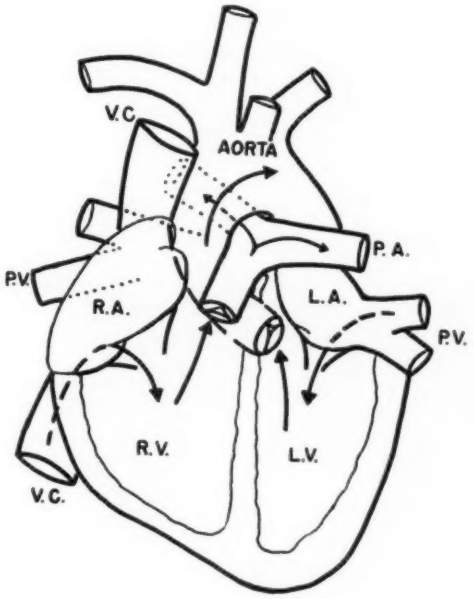
NORMAL

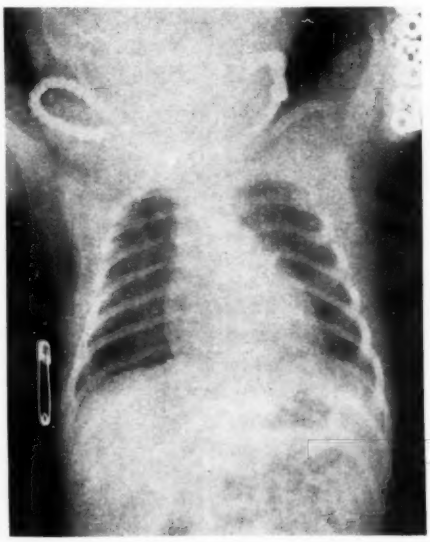
Clinical Features

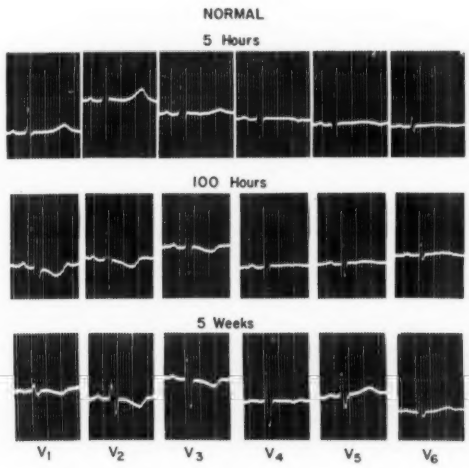
1. Cyanosis may be present at birth
2. Systolic murmur in early infancy
3. "Physiologic" right ventricular hypertrophy

Physiologic Data

1. Mean pulmonary artery pressure greater than *mean right ventricular* pressure
2. Mean right auricular pressure 4 to 7 cm. water
3. Absence of significant oxygen gradient between vena cava, right auricle, right ventricle and pulmonary artery







The determination in early infancy of whether the heart is normal or not may be exceedingly difficult. The exact nature of a defect when present may also be difficult unless all the methods outlined later are utilized. It may be stated here that for the determination of heart size, and particularly single chamber enlargement, the electrocardiogram is of more value than x-ray, normal criteria being available in the references cited.

AORTIC RING

Clinical Features

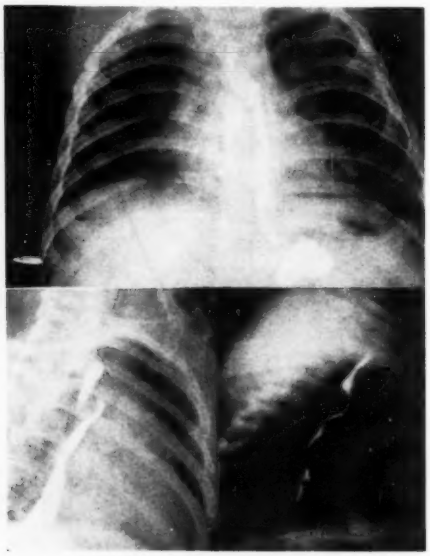
1. Regurgitation and aspiration
2. Respiratory stridor even when asleep

Physiologic Data

1. Normal

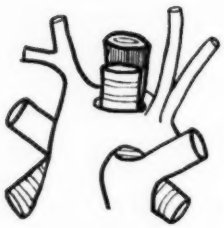
Conclusive Diagnosis

1. X-ray

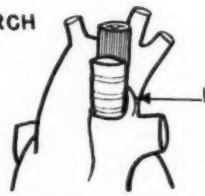


DIVIDED POSTERIOR AORTIC ARCH

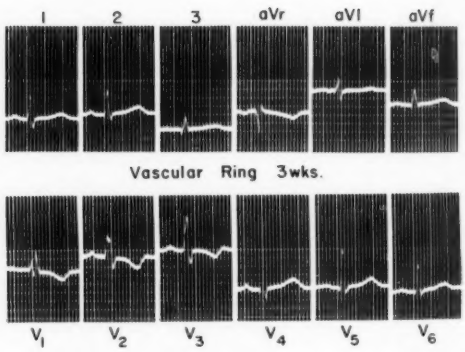
DIVIDED LIGAMENTUM ARTERIOSUM



DOUBLE AORTIC ARCH



RIGHT AORTIC ARCH AND
LEFT LIGAMENTUM ARTERIOSUM



The left posterolateral approach is used for all types of vascular rings. If there is a left aortic arch, the smaller component of the ring is divided (upper figure) or a retroesophageal right subclavian artery is divided. If there is a right aortic arch, the ligamentum arteriosum is divided (lower figure).

PATENT DUCTUS ARTERIOSUS

Clinical Features

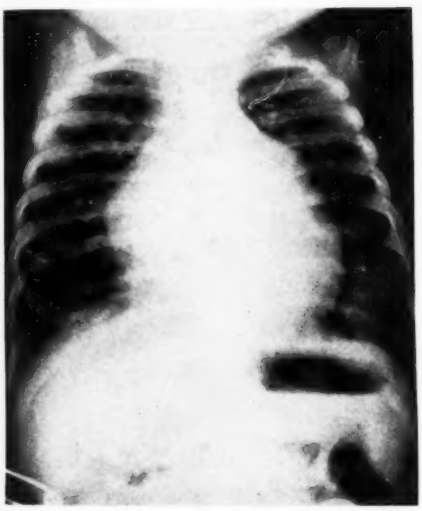
1. May have congestive failure early
2. Systolic murmur—absence of diastolic early
3. Wide pulse pressure
4. Normal heart size or left ventricular enlargement
5. Pulmonary congestion

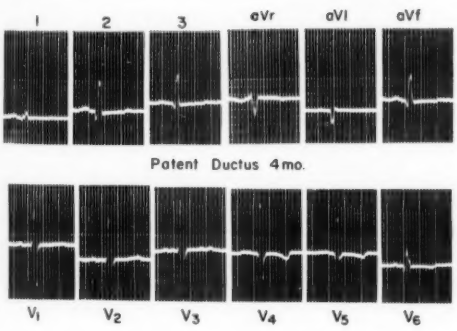
Physiologic Data

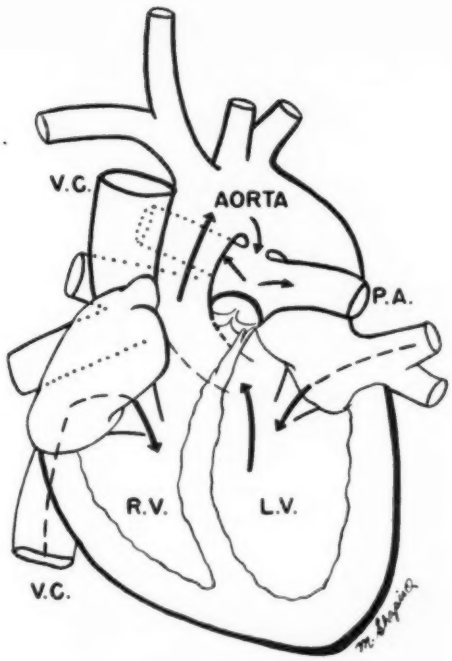
1. Pulmonary artery oxygen greater than right ventricular oxygen
2. Pulmonary pressure normal or increased
3. Direct catheterization of aorta through patent ductus

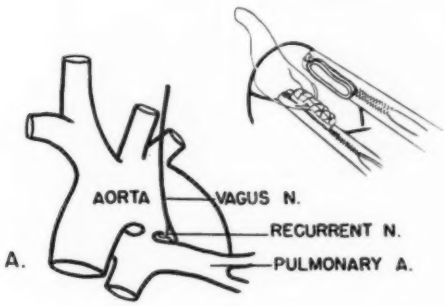
Conclusive Diagnosis

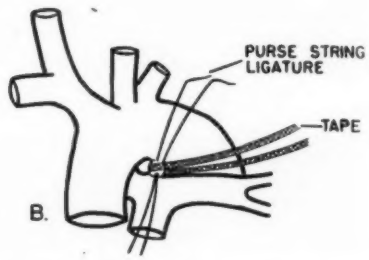
1. Cardiac catheterization
2. Retrograde angiography



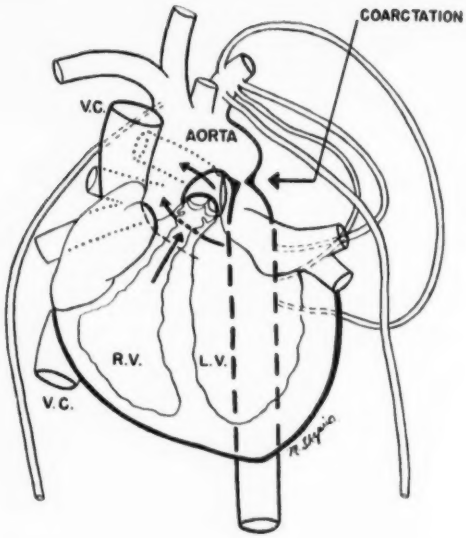








Through a left posterolateral incision, the ductus is exposed, care being taken not to injure the recurrent nerve. The ductus may be divided between the serrated ductus clamps of Potts and closed by suture (A, inset) or it may be triply ligated with two purse-string ligatures and umbilical tape (B).



COARCTATION OF THE AORTA

Clinical Features

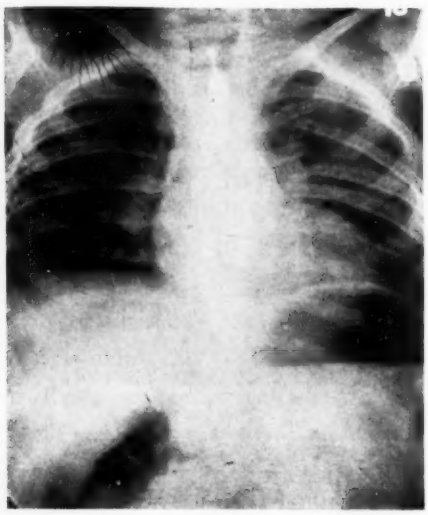
1. Right-sided cardiac failure early
2. Posterior transmission of murmur
3. Collateral circulation late

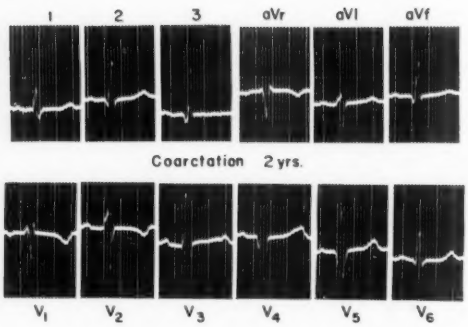
Physiologic Data

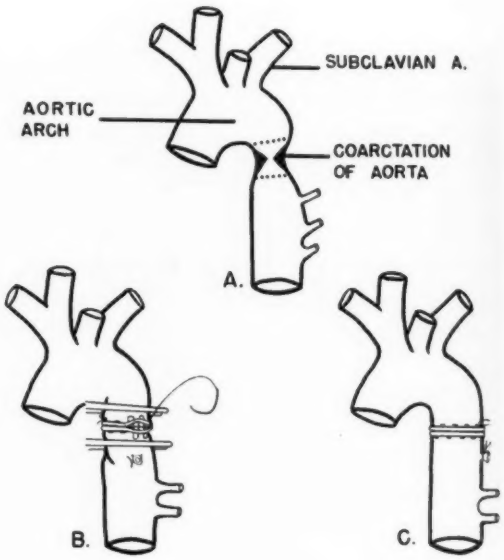
1. Normal if no associated defects

Conclusive Diagnosis

1. Blood pressure—upper and lower extremities
2. Retrograde angiography

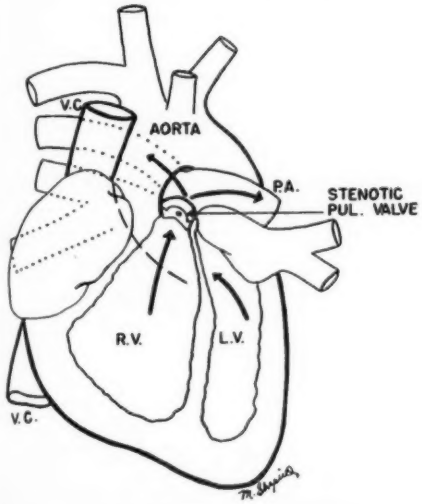


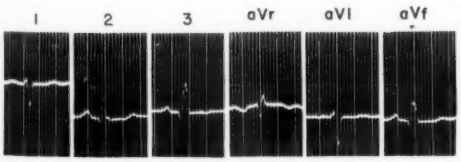




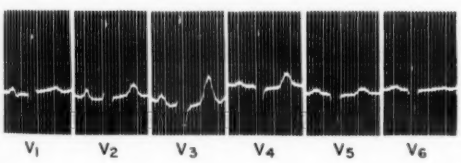
Through a posterolateral incision with transection of several ribs, the area of constriction is exposed, and the ligamentum arteriosum and one or more intercostal arteries are divided. After clamping with the Potts serrated clamps, the constriction is excised and continuity restored. Rarely, a homograft will be necessary.

LESIONS ASSOCIATED WITH CYANOSIS





Pulm. St. with Closed Septa 7mo.



PULMONARY STENOSIS WITH CLOSED SEPTUMS

Clinical Features

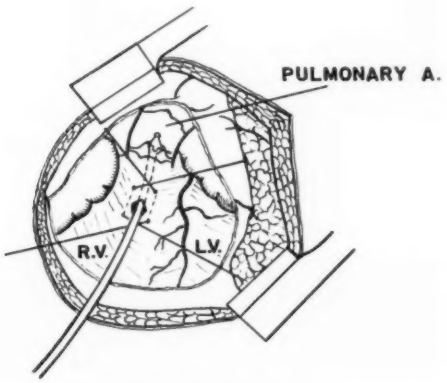
1. Large heart and cardiac failure (right-sided)
2. Clear lungs
3. No cyanosis
4. Marked right ventricular enlargement

Physiologic Data

1. Right ventricular pressure markedly increased
2. Low pulmonary artery pressure (if entered)
3. Right ventricular systolic pressure different from aortic systolic (femoral or brachial)
4. Normal right auricular and right ventricular oxygens
5. Prolongation of circulation time from the right ventricle
6. Femoral artery oxygen normal or only slightly decreased

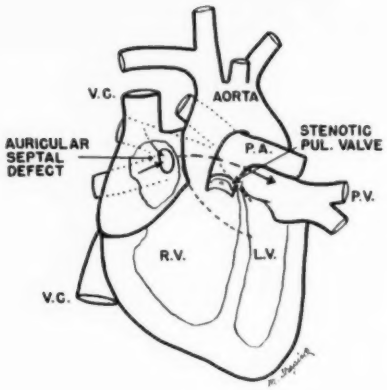
Conclusive Diagnosis

1. Clinical
2. Physiologic studies



BROCK OPERATION

Through an opening in the anterior surface of the right ventricle, first a uterine probe and then a Brock valvulotome of appropriate size are passed through the stenotic pulmonary valve.



PULMONARY STENOSIS WITH AURICULAR DEFECT OR PATENT FORAMEN OVALE

Clinical Features

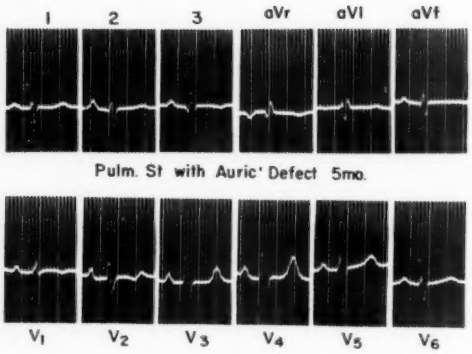
1. May closely resemble tetralogy
2. Cyanosis
3. Hyperpnea
4. Heart size variable, right ventricular hypertrophy
5. Clear lungs

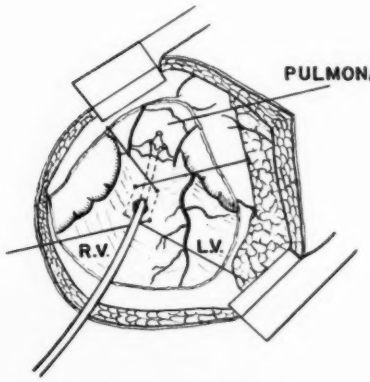
Physiologic Data

1. Right ventricular pressure markedly increased
2. Low pulmonary artery pressure
3. Right ventricular systolic pressure different from aortic systolic
4. Prolongation of circulation time from right ventricle
5. Significant oxygen unsaturation in left auricle and femoral artery

Conclusive Diagnosis

1. Clinical
2. Physiologic studies (including circulation time from right ventricle)

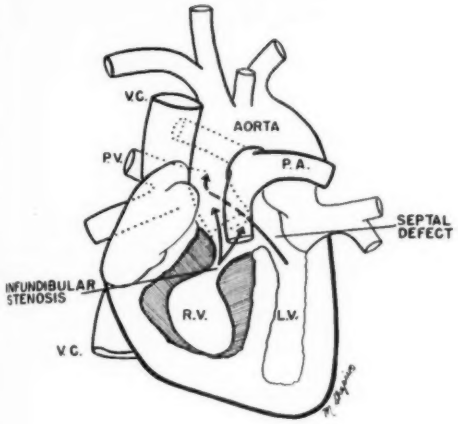


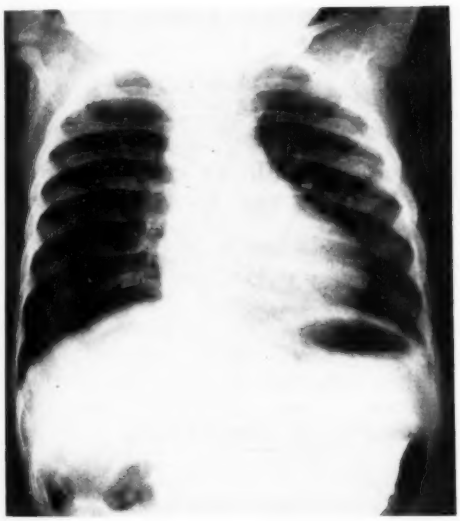


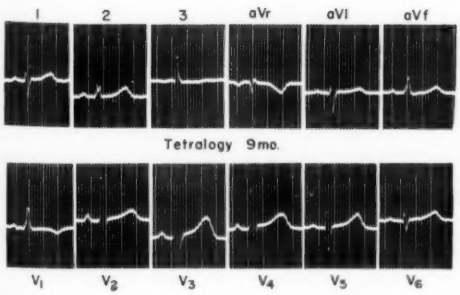
BROCK OPERATION

Through an opening in the anterior surface of the right ventricle, first a uterine probe and then a Brock valvulotome of appropriate size are passed through the stenotic pulmonary valve.

TETRALOGY OF FALLOT







Clinical Features

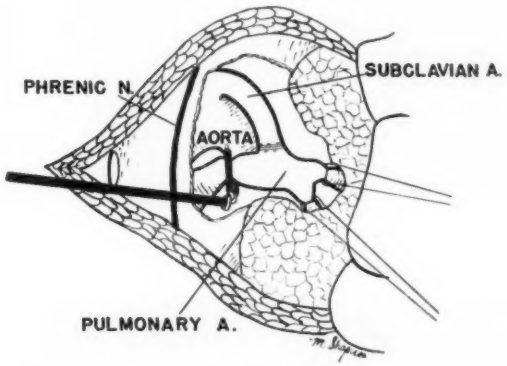
1. Cyanosis variable
2. Hyperpnea usually marked
3. "Spontaneous" attacks
4. Normal size heart; right ventricular hypertrophy
5. Clear lungs

Physiologic Data

1. Right ventricular pressure increased
2. Decreased pulmonary artery pressure
   Mean pulmonary artery less than mean right ventricular pressure
   Systolic pulmonary artery less than systolic right ventricular pressure
3. Right ventricular oxygen greater than right auricular oxygen
4. Direct catheterization of aorta from right ventricle
5. Short circulation time from right ventricle
6. Right ventricular systolic equal to aortic systolic pressure

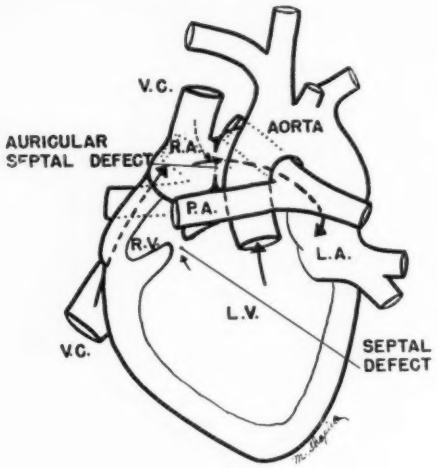
Conclusive Diagnosis

1. Clinical
2. Physiologic studies (including circulation time from right ventricle)



BLALOCK OPERATION

Some type of shunt must be made between the systemic and pulmonary circulations. A subclavian-pulmonary anastomosis may be indicated, particularly if the aortic arch is on the right, but in infants, the Potts operation (see page 150) is usually carried out.



TRICUSPID ATRESIA

Clinical Features

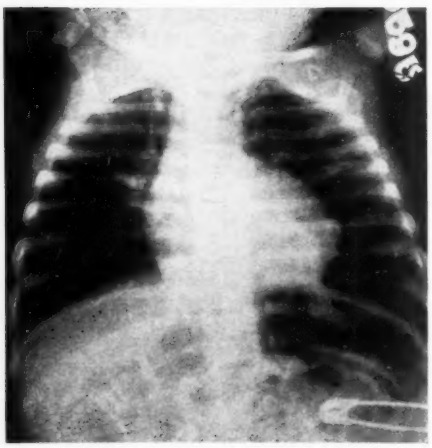
1. Cyanosis and hyperpnea
2. Left ventricular hypertrophy
3. Clear lungs

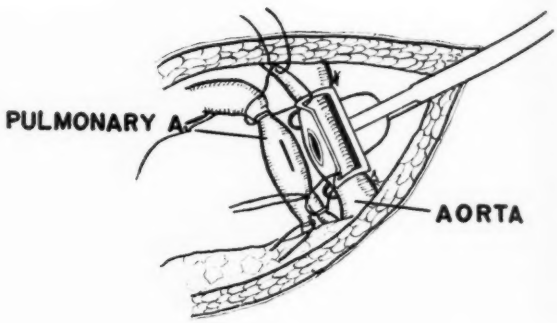
Physiologic Data

1. Inability to enter right ventricle

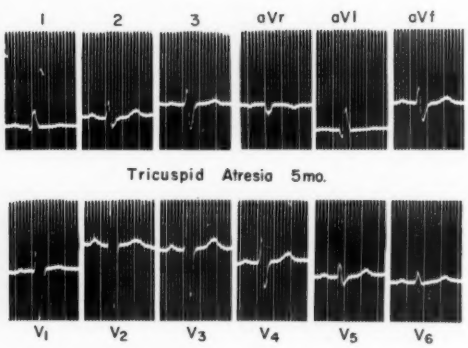
Conclusive Diagnosis

1. Clinical
2. Electrocardiogram



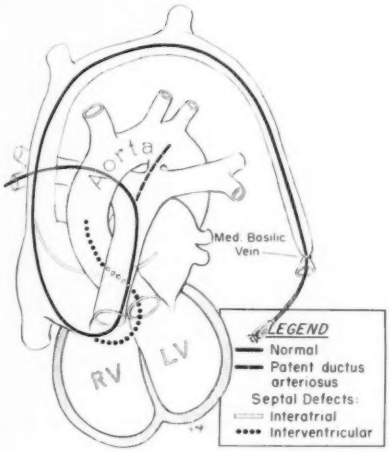


POTTS OPERATION



In infants with tricuspid atresia and an adequate interauricular defect, the Potts operation (aortic-pulmonary anastomosis) is usually beneficial. If the interauricular defect is small, a right subclavian-pulmonary anastomosis may be combined with the creation of a new interauricular defect (Blalock-Hanlon operation).

CARDIAC CATHETERIZATION

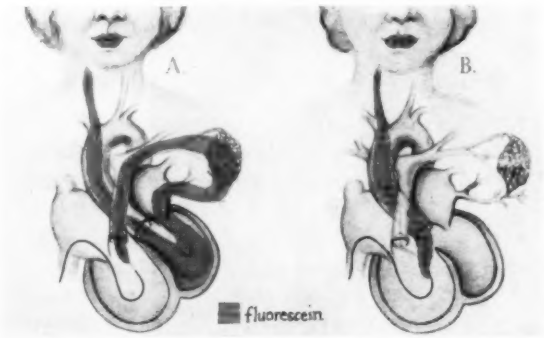


Possible pathways in cardiac catheterization. The position of the catheter is of diagnostic importance and, in addition, the following information may also be obtained: (1) intracardiac and intravascular pressure relationships, (2) oxygen content of blood from various positions and the determination of shunts, (3) right ventricle to face circulation time with fluorescein (see below) and (4) intracardiac electrocardiograms.

In infants and small children, catheterization may be done from the saphenous vein and inferior vena cava rather than from the arm as depicted in this illustration. No general anesthesia is necessary except in rare cases. Sedation with a combination of morphine and sodium phenobarbital is usually satisfactory, together with local infiltration with procaine 2 per cent. Complications in our experience, even in early infancy, have been rare, although experience and caution, combined with good clinical judgment, are priceless safeguards. For further information see references.

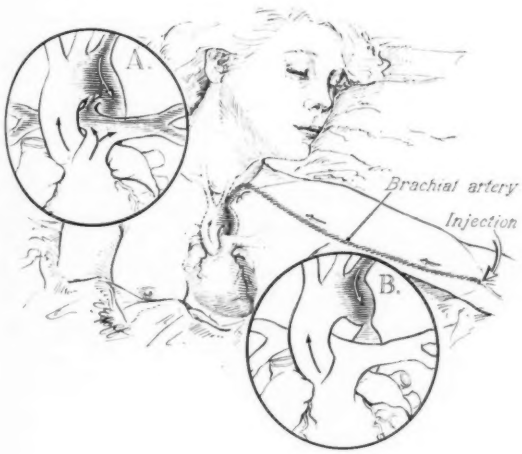
VENTRICLE TO FACE CIRCULATION TIME

With the catheter tip lying free in the outflow tract of the right ventricle, fluorescein is injected, and the time determined to the development of fluorescence of the lips under ultraviolet light.



A. Circulation time in valvular pulmonary stenosis with closed ventricular septum. Note that the dye must traverse the entire pulmonary circulation and left side of the heart before reaching the end point.

B. Circulation time in pulmonary stenosis and interventricular septal defect (tetralogy of Fallot). In this situation the dye can enter the aorta directly, thus appearing in the face much earlier than in A.

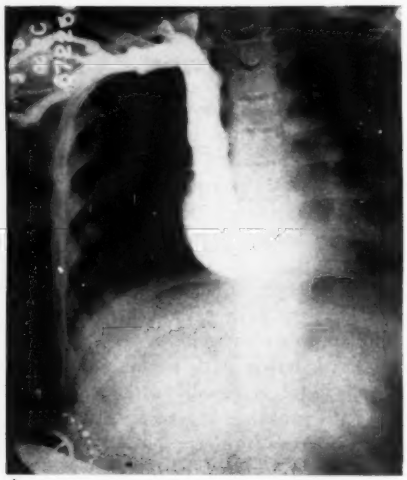


RETROGRADE AORTOGRAPHY

Radiopaque substance is injected in a retrograde fashion into the aortic arch via the left subclavian artery.

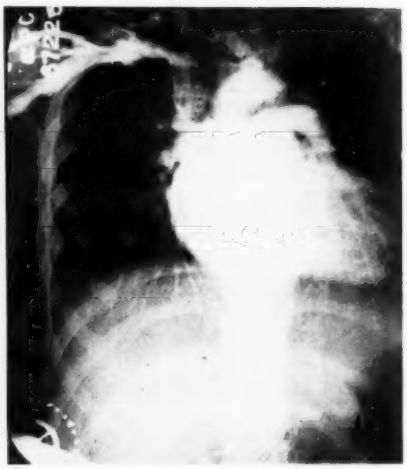
A. Patent ductus arteriosus. Note simultaneous filling of the pulmonary circulation and aorta.

B. Coarctation of the aorta.



A

B



C

ANGIOCARDIOGRAPHY IN A CASE OF TETRALOGY OF FALLOT

The films shown were taken at one-half second intervals.

A. Vena cava and right auricle.

B. Early opacification of right ventricle and pulmonary artery.

C. Simultaneous filling of aorta and pulmonary artery.

These films do not show the pulmonary stenosis which in this case was apparent clinically and proved by cardiac catheterization but they do show the simultaneous filling of the aorta and pulmonary artery characteristic of a high interventricular septal defect and dextroposition of the aorta.

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Streptococcal Diseases of Childhood

FLOYD W. DENNY, JR.*

University of Minnesota Medical School, Minneapolis

STREPTOCOCCAL respiratory infections and their suppurative complications are a major cause of illness, particularly in children. The subsequent occurrence of acute rheumatic fever in 3 to 5 per cent of patients with streptococcal diseases greatly increases the importance of this group of infections. It is now possible by use of the antibiotics to shorten the course of streptococcal diseases, to eradicate the streptococci and to prevent the subsequent occurrence of acute rheumatic fever. In order to accomplish these things effectively the infections must be recognized early and adequate treatment instituted immediately. The purpose of this paper is to describe the clinical and laboratory manifestations and the treatment of streptococcal infections in children.

Streptococci

The *Streptococcus* responsible for respiratory infections and rheumatic fever is classified as beta hemolytic because of its ability to produce complete hemolysis on a blood agar culture plate. Beta hemolytic streptococci can be differentiated into groups which are designated by letters of the alphabet. Those in group A cause disease in man; other groups cause disease in animals and are found occasionally in man as harmless parasites. Group A beta hemolytic streptococci may be further differentiated into serologic types which are designated by numbers. There is evidence that type 12 infections may be followed by glomerulonephritis more commonly than other types,[1] but with this exception there is nothing to indicate that different types possess different disease-producing capacities.

The group A beta hemolytic *Streptococcus* is composed of several antigenic components that excite the formation of specific antibodies which can be measured in the laboratory. The presence of these antibodies is of great importance in the study of streptococcal diseases and rheumatic fever, but their measurement is not routinely necessary for the adequate diagnosis and treatment of streptococcal infections.

The beta hemolytic *Streptococcus* may be isolated from the throats of 5 to 10 per cent

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of the children in the normal population. In these individuals the organism seems to be causing no harm, but it may indicate the presence of or recovery from a subclinical streptococcal infection.

Clinical Manifestations

Invasion of the upper respiratory tract by group A streptococci is followed by a disease that differs with the age of the patient.[2] The very young child, under 6 months of age, may either be asymptomatic or have a brief illness characterized by irregular fever, thin nasal discharge, slight pharyngeal infection and only mild systemic reaction. From this age until about 3 years another type of illness is usually seen. The patient is much more ill and may remain so for several weeks. The onset is insidious with fever, red pharynx, vomiting, anorexia and cervical adenopathy. Suppurative complications are common but rheumatic fever is rare. The important fact about streptococcal disease at this age is that it seems to be a subacute, generalized and protracted illness.

After the age of 3 years the *Streptococcus* causes a short, stormy, well localized illness that has commonly been called a "strep throat." These patients have the sudden onset of chills, fever, headache and a sore throat that usually makes swallowing painful. The tonsils and pharynx are quite red and may be covered with a creamy exudate. There is marked tonsillar enlargement and lymphoid hyperplasia of the pharynx, and cervical lymph nodes are large and tender. In spite of this rather severe reaction the patients are usually much improved or quite well within a few days. Suppurative complications are more rare than in the earlier age groups but the subsequent development of rheumatic fever is much more common. It is in this age group that scarlet fever most frequently occurs.

In spite of the severity of some streptococcal infections it should be stressed that a large number produce atypical symptoms or are asymptomatic. For this reason it is important and necessary in many cases to make or confirm the diagnosis by laboratory methods.

Lymphadenitis, otitis media, sinusitis, pneumonia, empyema, peritonitis, osteomyelitis, cellulitis, endometritis and vaginitis can be caused by beta hemolytic streptococci but usually occur as complications of infections of the respiratory tract.

Laboratory Findings

The peripheral leukocyte count is usually elevated in streptococcal infections, counts of 12,000 to 20,000 per cubic millimeter of blood being common. This observation is extremely useful in differentiating streptococcal infections from cases of the common cold or of nonstreptococcal exudative pharyngitis, with which they are so easily confused. Low or normal peripheral leukocyte counts characterize these two illnesses.

The demonstration of large numbers of beta hemolytic streptococci on blood agar culture plates is very helpful in making a diagnosis. While it is true that some normal people are carriers of group A beta hemolytic streptococci, or nonpathogenic hemolytic streptococci of other groups, the occurrence is so infrequent that it does not materially detract from this method of diagnosis. The number of streptococcal colonies is of some importance; in cultures from carriers the number is usually small while in cultures from infected individuals the number is large.

The recommended technic of making a throat culture to demonstrate streptococci is as follows: A dry, sterile, cotton-tipped swab is passed vigorously several times over the pharynx and both tonsils. It is then immediately inoculated onto the surface of a previously warmed blood agar plate. After 18 to 24 hours' incubation at 37° C. the plate is read for the presence of colonies with clear hemolysis which are shown in smears to be composed of gram-positive cocci, usually but not always in chains. Colonies of the hemolytic *Staphylococcus* are easily distinguished from the *Streptococcus* by their large size, mucoid consistency and chalky white or yellow color. It has been shown that if sheep's blood is used in the blood agar plates the growth of hemolytic colonies of the *Hemophilus* series of organisms and of many nongroup A streptococci is inhibited.[3]

The demonstration of a rise in streptococcal antibodies following a streptococcal infection is the only method at the present time of making a positive, unequivocal diagnosis. This method is, however, of little use clinically for several reasons: The methods for measuring these antibodies are not adaptable to routine laboratory use; the appearance of antibody is not detectable until at least 10 days after the onset of the infection, and if the streptococcal infection has been adequately treated the formation of antibodies is markedly or completely suppressed.

Treatment

Penicillin, AUREOMYCIN® and terramycin are equally effective in relieving the symptoms and signs of streptococcal pharyngitis and tonsillitis and also in the degree to which they suppress the development of streptococcal antibodies.[4] Penicillin is slightly more effective than the other two drugs in the speed and degree of eradication of the *Streptococcus*. All three drugs are highly effective in preventing suppurative complications.

Aureomycin and terramycin are usually administered by mouth and penicillin by the intramuscular route. There is little information concerning the use of penicillin by mouth in the treatment of streptococcal infections but there is no reason to believe that it would not be effective if given in adequate doses by that route. Full therapeutic doses of these drugs should always be used over a period of *at least seven days and if possible 10 days.* Shorter periods of treatment are inadequate and lead to a high percentage of recurrences.

The sulfonamides have been shown to be much less effective than antibiotics in the treatment of streptococcal respiratory infections and for this reason they are not recommended.

Prevention of Rheumatic Fever

It is now well established that if streptococcal infections are treated early and adequately with penicillin[5] or aureomycin,[6] rheumatic fever is prevented in the great majority of cases. Penicillin is probably slightly more effective than aureomycin in this respect. No figures are available on the prevention of rheumatic fever with terramycin. The sulfonamides are ineffective when used for this purpose.[7]

Prophylaxis of Streptococcal Infections

In patients who have had rheumatic fever the recurrence rate is extremely high following a new streptococcal infection. Since the prevention of rheumatic fever by treatment of these infections is not always successful and since streptococcal infections are not always recognized, it is important to prevent the occurrence of these illnesses. At the present time there are two agents that are effective when used for this purpose. Sulfadiazine, in contradistinction to its relative ineffectiveness in the treatment of streptococcal infections, is very effective in preventing the occurrence of streptococcal infections.[8] One-half gram once or twice a day is the recommended dose. Oral penicillin in doses of 100,000 units two or three times a day, 30 minutes before meals, is also effective in preventing the occurrence of streptococcal infections.[9] Prophylaxis with either of these drugs should be continued without interruption for at least two years and if possible five years after the initial attack of rheumatic fever. The patients should, of course, be carefully observed for the development of toxic reactions.

Summary

The group A beta hemolytic *Streptococcus* is a common cause of disease in children. The proper diagnosis of the various types of illnesses caused by the *Streptococcus* is at times difficult and is best accomplished by combining clinical and laboratory observations. Early and adequate therapy with penicillin, aureomycin and terramycin relieves the symptoms and signs of streptococcal infections and reduces the number of suppurative complications. Penicillin and aureomycin are effective in preventing the occurrence of rheumatic fever. In patients who already have rheumatic fever it is desirable to prevent the occurrence of streptococcal infections by the prophylactic use of sulfadiazine or penicillin.

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Home Care for Acute Poliomyelitis

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In the last year or two the importance and practical advantages of adopting home care for patients suffering from one or another of the milder forms of acute poliomyelitis have attracted wide recognition and pretty general acceptance. There are good reasons for keeping such patients at home.

P. M. STIMSON

In the first place, fatigue and physical exhaustion just before or after the clinical onset of acute poliomyelitis are known to be definitely detrimental to the patient's welfare, in some cases probably markedly increasing the severity of muscle involvement. Keeping the patient quietly in his own bed at home and avoiding the fatigue and nervous tension incident to transportation and to the first days in a hospital are in line with the dictum that rest, both physical and psychologic, is the most important factor in the early care of patients with this frightening disease. Furthermore, to stay at home is naturally the patient's preference, and usually that of his family, and danger of infection to the others in the home is not appreciably increased, the patient probably already having exposed and infected all his contacts for some days before the diagnosis is suspected.

The cost of the illness with home care is naturally much less to the family or to assisting agencies than would be the case with hospital care, and with the patient at home the trusted family doctor can usually continue in attendance. From the hospital viewpoint, keeping patients with mild cases at home will not only release much-needed hospital beds and nurses for the more serious cases but also frequently prevent admission to hospitals of many patients with trivial illnesses that are only suspected of being poliomyelitis.

Thus if a patient newly ill with this disease can safely be kept at home, the reasons for doing so are strong, but there must be a careful determination as to which patients may be kept at home and which should be sent to a hospital.

Those kept quietly at home may include patients who are only "suspect" and not particularly ill, especially the ailing siblings in

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a family of a patient with a recognized case. Patients more definitely sick, with fever, headache, a scratchy throat, perhaps pains and stiffness in neck, back and extremities, but no recognizable weakness, and who are considered to be possible instances of acute poliomyelitis without paralysis as yet, might well be kept quietly where they are and carefully watched for possible progression of the infection. Even when some weakness appears in an extremity to make the diagnosis quite certain, there is really nothing that hospitals can do for mild paralytic cases in the acute stage that good homes cannot do. Unfortunately, in many hospitals there too often are injudicious treatments and excessive handling, and as a result many a patient might be better off if he were at home.

However, a lumbar puncture will often be wanted for added information, particularly to rule out a beginning meningitis, but it must be remembered that a normal spinal fluid by no means eliminates a diagnosis of poliomyelitis, nor does a mild increase of mononuclear cells mean more than that there is present some kind of inflammation of the central nervous system. Health authorities in each community should make it possible that diagnostic lumbar punctures, when indicated, can be provided in homes. But unless there is a chance of the presence of a meningitis, the lumbar puncture may often be omitted, or at least postponed, particularly if the patient's illness is not progressing.

Of course, the home should have adequate facilities of accommodations and personnel, and the community should be able to provide medical consultations to back up the family doctor, as well as nursing services and, later, physical therapists and perhaps certain special equipment.

On the other hand, there are indications of trouble, present or impending, which when found in a sick person demand his hospitalization if it is in any way possible, and of course preferably in a hospital where there is real knowledge of the proper care of poliomyelitis. These indications include increasing fever; an increasing appearance of sickness and prostration; difficulties of urination; weakness of a deltoid muscle, because this is apt to be associated with diaphragmatic weakness; any breathing difficulty even though it be only an increase in restlessness and in the rate of respiration; a change in the character of the voice; regurgitation of fluids through the nose or difficulty in swallowing, or a marked increase in blood pressure, which might mean an early irritation of the vasomotor center and possible later destruction of that center.

The decision to send a patient to a hospital may be strengthened by the fact that not only is the home inadequately equipped for caring for a patient, but also there may be too much emotional instability in the family and too much panic in the neighborhood. Furthermore, busy doctors like to concentrate their patients in a hospital rather than having to watch a number of them scattered in their homes. If a transfer to a hospital is made, it should be done with a minimum of fatigue to the patient.

Treatment in the home of nonparalytic patients and even of those with slight weaknesses is not complicated, particularly during the acute stage while there is fever. The essential factor is rest and peace—of body and mind. While patients with backs that are tight and sore need a bed made flat and firm by a bed board, those with mild cases need only a reasonably firm mattress and bed, and they should be allowed to rest in any position in which they are most comfortable. Isolation requirements should be met as well as possible and public health nurses should be available for teaching the families how best this can be accomplished. The patient should of course have a bed to himself and preferably a separate room, and those in attendance should wash their hands carefully after each contact with the patient.

Sleep is the best therapeutic measure for early acute poliomyelitis and should be interrupted for no reason whatever. Sedation is dangerous, however, for if the disease spreads and breathing difficulty develops during enforced sleep, there is danger of the breathing stopping. Being read aloud to is an excellent soporific for many children and the admin-

istration of acetylsalicylic acid is permitted.

Backaches and general tenderness can best be relieved by moist heat, most advantageously given in many homes by immersion in a bathtub with water at body temperature, subsequently raised to 102 to 104° F. Because of the possibility of the patient's becoming light-headed and even fainting while in the hot water, he should never be left alone in the tub. Cold applications to the head help to prevent this light-headedness. Two to four such soaks a day, each lasting 5 to 10 minutes, are soothing and comforting, though exhausting to some. When use of a tub is not practical, if the patient in bed is gently turned onto his face, hot packs made of pieces of wool blanket dipped in boiling water, then put twice through a wringer and gently placed on the patient's neck, back, thighs, calves and feet and left on for 10 minutes, are both soothing and relaxing to tight, sore muscles. After such an application has been repeated once, if the patient is asleep, as is often the case, he should not be awakened, but if awake he may be turned onto his back and his extremities so moved that each joint is passively carried through its entire range of motion, but always only as far as can be done without causing appreciable pain. Such a treatment of packs and passive motion may well be given three or four times in 24 hours, especially just before a nap or before bedtime.

Except possibly for quinine which helps to relieve muscle twitchings and cramps in the voluntary muscles, and for aspirin which usually makes the patient generally more comfortable, there is little indication for other medication in the home care of acute poliomyelitis. Cathartics and laxatives are apt to cause only increased peristalsis without actual elimination, and simple enemas given every day or two are often necessary for a week or more. Slight difficulties in urination in nonparalytic patients, especially boys, can sometimes be overcome by allowing the patient to sit on the side of the bed or on a bedside commode in order to urinate.

Antibiotics are of no value in acute poliomyelitis except under three circumstances: (1) when there is breathing difficulty and the danger of a complicating lung infection; (2) when catheterization is necessary, and (3) when a high leukocyte count suggests the presence of a complicating bacterial infection. Cortisone is harmful.

After the first week or 10 days, complete muscle testing should be done. If no weakness can be found and the pain and stiffness are essentially gone, the patient can be allowed gradually progressive activity—first to a bedside chair, then to bathroom privileges and then to sedentary occupations—but a return to normal activities (still without free exercising) should take at least four weeks from the onset in any case considered to be a poliomyelitis virus infection.

To serve the family physician who feels he may need diagnostic help, advice in treatment or moral support, some county medical societies provide a panel of consultants. Public health nurses are also usually available for assisting in home care of the patient and instruction of the family. Communities should also be equipped with physical therapy services that can be called on when needed.

Campaigns of education are needed (1) to advance the idea of home care for patients with acute polio; (2) to teach doctors and families how to make home care practical and adequate, and (3) to convince communities and social agencies that it is far wiser to spend money not on the hospital care of inconsequential illnesses but rather on providing consultants for family doctors, visiting nurses for home care and instruction, follow-up facilities of physiotherapy, and rehabilitation clinics.

Summary

There are good reasons why home care should be used for more of the mild cases of acute poliomyelitis than has been customary, and clear indications for determining which patients should be sent to a suitable hospital. In a suspected case of poliomyelitis, the principal value of the examination of the spinal fluid is to rule out the presence of a meningitis. Proper home care of patients with mild cases is neither difficult nor complicated. Much educational preparation is necessary for spreading the practice of home care.

GENERAL PRACTICE *Review*

This department offers the latest, proved methods for the treatment and management of commonly encountered conditions based on the extensive experience of the physicians who prepare these summaries especially for POSTGRADUATE MEDICINE.

Medical Treatment of Massive Hemorrhage from Peptic Ulcer

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A PATIENT who is suddenly overtaken by a massive hemorrhage from a peptic ulcer immediately presents his physician with one of the most dramatic and urgent of all medical emergencies. Aaron, et al.[1] have stated that 80 to 85 per cent of all cases of massive upper gastrointestinal bleeding are due to peptic ulcer. Carcinoma of the stomach rarely causes massive hemorrhage. Other common causes are erosive hemorrhagic gastritis, hypertrophic gastritis, acute erosions associated with an alcoholic gastritis, hiatus hernias and ulceration or varices in the lower esophagus. In a series of cases of hematemesis reported by Cates,[2] 48 per cent were due to peptic ulcer, 3.4 per cent to carcinoma and 32 per cent to esophageal varices.

J. ALLEN WILSON

The mortality rate due to bleeding from peptic ulcer has been variously reported, by many authors, to be from 2.7 to 30 per cent. Several important factors in this mortality rate have been reported by Aaron, et al.[1] as follows: (1) Massive hemorrhage in older age groups is accompanied by a much higher mortality rate. (2) Hematemesis has a much more ominous significance than melena, meaning sudden and massive expulsion of blood into the upper gastrointestinal tract. (3) Recurrent hemorrhage while the patient is still in the hospital under observation is very serious. (4) The prognosis is more serious if additional disease is present (portal cirrhosis, essential hypertension or cardiac failure).

Several authors have stressed the higher mortality rate from bleeding gastric ulcers than from bleeding duodenal ulcers (often a ratio of 2 to 1). Pollard and Wollum[3] wrote that this is due to frequent erosion of the right or left gastric artery. These authors also pointed out the higher mortality rate from bleeding peptic ulcer in men as compared with women (2 to 1) in their series. The highest mortality rate was in those patients having symptoms of ulcer but negative

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roentgenographic findings. They also found, as have other authors, that most deaths from bleeding ulcer occurred with the first hemorrhage. The mortality rate was lower with succeeding hemorrhages. They also found that the mortality rate of patients having hematemesis was twice that of patients with melena only. The mortality rate was higher for those patients who bled in spite of a full medical ulcer regimen than for those not so treated.

Establishing the Diagnosis

When a patient has a massive upper gastrointestinal hemorrhage, it is urgent that the source of the bleeding be localized as soon as possible. Gray[4] has stated that a history of ulcer symptoms preceding the hemorrhage with the sudden cessation of all pain after the onset of bleeding justifies a tentative diagnosis of bleeding peptic ulcer. Statistically, previous episodes of hematemesis or of melena suggest the probability of peptic ulcer. Known roentgenographic findings are helpful in establishing the diagnosis. Esophageal varices, gastric carcinoma, gastric polyps, diaphragmatic hernia, gastritis and gastric erosions and blood dyscrasias must all be considered in the differential diagnosis. Esophageal varices are suggested by spider angiomas, a palpable firm liver or spleen, icterus and an alcoholic history. It is my rule to have a prompt determination of the BROMSULPHALEIN® dye retention test on any patient who has massive upper gastrointestinal bleeding. If it is elevated markedly above 8 to 10 per cent retention of dye, this test gives strong evidence of bleeding from esophageal varices.

If the diagnosis of the site of bleeding is uncertain, and bleeding continues in spite of treatment, a cautious roentgenographic examination of the esophagus, stomach and duodenum should be done by Hampton's[5] method—a profile, relief and double contrast examination of the esophagus, posterior wall of the stomach, pyloric valve and posterior wall of the duodenum with the patient in the horizontal position and without compression or manipulation. If the site of bleeding is known, roentgenographic examination should be postponed until the bleeding has stopped and compression and manipulation can safely be utilized.

A few laboratory tests should be made when the patient is first seen. These include the blood type, Rh factor, and cross matching with suitable blood in adequate amount. The hemoglobin, red blood cell and hematocrit determinations must be done at once and repeated at frequent intervals in the face of continuing hemorrhage (at least every four to six hours in the first few days of management). These are apt to be normal or nearly normal in the first few hours after an initial hemorrhage. The blood pressure, of course, is the most valuable single test and should be repeated every 15 to 30 minutes if there is continuing hemorrhage. The prothrombin time should be determined. It is high with liver disease or in anoxia of the liver due to severe hemorrhage. All stools and vomitus must be examined for gross and occult blood. Occult blood is especially important in determining whether bleeding has finally ceased. The bromsulphalein liver function test should be done promptly.

If there is a history of vomiting or alkali use prior to the hemorrhage, determination of the blood carbon dioxide and chlorides is valuable. As Gray[4] mentioned, a blood urea nitrogen determination may be helpful, for it often can be used as an index of the severity of the hemorrhage into the gastrointestinal tract and of alimentary absorption of digested blood. Bockus[6] also quoted several authors' experiments to show that 500 to 600 cc. of blood in the human gastrointestinal tract may double the normal blood urea nitrogen level within eight hours and that the maximum rise in the blood urea nitrogen level occurs from 24 to 48 hours after the blood enters the intestine. However, a rapid fall of the blood urea nitrogen level to normal within three to four days after the cessation of bleeding can be expected if shock or impaired renal function has not occurred. This alimentary hyperazotemia may be accompanied by some fever and toxic symptoms between the second and fifth days after the onset of bleeding. A good enema may relieve these symptoms rapidly. Shock and dehydra-

tion produce impairment of renal function and may elevate the blood urea nitrogen level. Bockus[6] said that the blood urea nitrogen level together with the blood counts, hematocrit reading, blood pressure and condition of the circulation suffice to establish or disprove continued or renewed bleeding in most cases. Extremely high blood urea nitrogen levels, according to Bockus, are rarely due to absorption of blood alone. Blood urea nitrogen levels of over 100 mg. per cent offer a very grave prognosis.

Blood Transfusions

The one most important means we have to combat the dangers of massive hemorrhage from peptic ulcer (as also from other causes) is the judicious use of blood transfusions. Obviously transfusions are not needed nor wisely used in patients in whom the hemorrhage is not severe. Pollard and Wollum[3] stated that the fall in blood pressure if not too great may actually be of benefit in preventing further hemorrhage. Blood clots more rapidly after a not too severe hemorrhage and this, combined with a moderate lowering of blood pressure, helps to seal the leak even in fairly large vessels. In the case of arteries, retraction of the middle fibromuscular coat and curling of the endothelial lining are further defenses against bleeding. These authors said that the role of therapy in hemorrhage from peptic ulcer consists first in the cautious withholding of transfusions at the onset of bleeding. This allows the described mechanisms to effect cessation of hemorrhage in the majority of cases. However, if the systolic blood pressure drops to 90 mm. of mercury or if the hemoglobin falls to 50 per cent Pollard and Wollum immediately begin the infusion of 500 cc. of citrated blood by slow drip over a three to four hour period, repeating often enough to control shock and anemia. These authors also emphasized, as did Emerson and Ebert,[7] that the hemoglobin, red blood cell count and hematocrit readings lag behind the clinical course of the patient. A falling blood pressure is the most important guide to impending shock.

Emerson and Ebert[7] pointed out, from careful studies on shock following blood loss in battle casualties, that the shock syndrome is essentially a reflection of a diminished blood volume due to hemorrhage. Blood volume studies are the most reliable method of evaluating the likelihood or presence and degree of shock, but such methods are not available to the average clinician. Emerson and Ebert further stated that the ideal shock therapy entails restoration of blood volume to near normal levels and of hemoglobin concentration to adequate levels. The hemoglobin level lags behind falling blood volume and therefore is not an adequate guide to the need for transfusions. They emphasized that the level of arterial blood pressure is the only reliable clinical criterion of the need for transfusion. Patients with a systolic blood pressure consistently below 85 mm. of mercury had a blood volume deficit between 25 and 40 per cent, while those with pressures over 85 mm. of mercury had less than 25 per cent deficiency in blood volume.

In consideration of this excellent work of Emerson and Ebert, in cases of massive hemorrhage I would suggest the following transfusion plan:

1. In patients who have had a massive melena or hematemesis but whose systolic blood pressure is near normal (over 85 to 90), 1000 cc. of citrated blood should be given by slow intravenous drip over a period of six hours. If the pressure is over 100 mm. of mercury, one transfusion of 500 cc. of blood may suffice.

2. If the arterial blood pressure is below 85 to 90 mm. of mercury, 1500 to 2000 cc. of blood should be given in the first 6 to 12 hours, the rate of infusion depending on the course of the blood pressure which should be determined every 15 to 30 minutes.

3. If the blood pressure does not respond and rise to 100 mm. of mercury or over and the patient is evidently losing more blood by vomiting or through the bowel, several more pints of blood should be available and the blood infused rapidly, preparatory to surgical therapy if it is needed. Emerson and Ebert[7] recommend that in patients being prepared for surgery 1000 cc. more blood be given

beyond the amount needed to restore the arterial pressure to 100 mm. of mercury. Aaron[1] has stated that up to 2000 cc. of blood can safely be administered in one hour when there is continuous heavy blood loss.

4. During major surgery, 2000 to 3000 cc. more blood may be needed to restore the blood volume loss resulting from and during surgery.

5. If the arterial pressure is well restored by the transfusions, and emergency surgery is not deemed necessary, the blood pressure, hemoglobin and hematocrit determinations should be repeated frequently (especially the blood pressure readings) and further blood may be withheld unless new hemorrhage and decrease of blood volume are in evidence.

6. In older patients with cardiac disease, one important way to avoid overloading the circulation (after the first few hours of hemorrhage) is to give one or two bottles of whole blood followed by further transfusions of blood in which the red cells are concentrated by discarding one-third to nearly one-half of the plasma in each unit of blood.

To Stop Bleeding

Several means of attempting to bring about the cessation of bleeding from the stomach and duodenum are available. Cantor, et al.[8] have had excellent results from using 4 tablespoons of GELFOAM® powder (dry) every few minutes for three or four doses and then giving 2 tablespoons of this powder every two hours, day and night for three to five days, followed by 250 units of bovine thrombin dissolved in 50 cc. of water or saline. One tablespoon of AMPHOJEL® is also given every three hours for its antacid action and to reduce the digestive action of pepsin on the blood clot present at the site of hemorrhage. Excellent results were reported in a series of 120 patients ranging in age from 18 to 89 years.

Sengstaken and Blakemore[9] have devised a nasogastric tube with a double balloon which can exert effective pressure on bleeding esophageal varices (or esophageal peptic ulcers), but which also can be used effectively against bleeding coronary veins in the gastric wall and conceivably might be useful in checking bleeding from high-lying gastric ulcers on the lesser curvature or posterior wall near the cardia.

Vitamin K may check the bleeding tendency where anoxia of the liver following massive hemorrhage has reduced the prothrombin production of that organ. Dr. B. W. Sippy years ago occasionally stopped a severe hemorrhage by lavaging the stomach with ice water (probably effective due to splanchnic vasoconstriction produced temporarily) and then just before the Ewald tube was withdrawn he instilled 1 ounce of 1:1000 ADRENALIN® solution into the stomach for its vasoconstrictor action on the mucosa. I was able to stop continuing massive hemorrhage by this method in 1 patient.

Therapy

Once the hemorrhage is under control, the patient should have a daily fluid intake of 2000 to 2500 cc. depending on his state of dehydration, azotemia, renal output and cardiac status. Intravenous fluids should be given slowly, especially to patients with cardiac disease. The latter usually should be given 5 or 10 per cent glucose in distilled water rather than in saline. A minimum urine output of at least 800 cc. daily should be effected by such parenteral and oral fluid administration. Bockus[6] emphasized that a progressive fall in the volume of the patient's urinary output in spite of adequate parenteral fluid is indicative of marked renal impairment and is a bad prognostic sign.

Unless the patient is vomiting repeatedly the diet should be started at once, even during evidence of active hemorrhage. Gray[4] wrote that small hourly feedings maintain the nutritional state of the patient and reduce gastric acidity. Mortality statistics are lower with this regimen than when the traditional 48 to 72 hours of fasting are observed. If the patient is in shock, however, 24 or, in rare cases, 48 hours of fasting may be needed until he improves sufficiently to tolerate hourly feedings of 100 cc. of milk and cream. No further additions of food are allowed for the first five to seven days, until it is certain that all bleeding has ceased and that the

stools show no occult blood reaction. Then small bland feedings may be added to the hourly milk and cream as the usual Sippy-type diet regimen. I feel, as does Gray,[4] that the Meulengracht diet has the disadvantage of increasing the vigor and amplitude of gastric contractions and of overstimulating large volumes of gastric acid.

In addition to the hourly feedings of milk and cream (used only till 7 P.M. daily) my antacid regimen is to give hourly doses (halfway between the milk and cream feedings) of ½ ounce of amphojel with magnesium trisilicate, or of GELUSIL®, throughout the day and then at hourly intervals during the evening to midnight and at two hour intervals from midnight till morning. The patient can be given a barbiturate in the evening so he only partially arouses for these night feedings. It has been my experience that this regimen is as effective in stopping hemorrhage as is the use of a continuous drip of aluminum hydroxide or of milk and antacid, and is technically much easier to carry out than the drip method. The possibility of causing a bowel obstruction by producing a firm mass of blood clot and aluminum hydroxide has been described. One such case is known to me but it has never occurred in my own experience.

During the period of bleeding, the patient should have absolute physical rest and complete mental rest. Barbiturates are the best medication for this dual purpose. LUMINAL SODIUM® by hypodermic injection every four to six hours may be used first. Later, NEMBUTAL® at bedtime and AMYTAL® or phenobarbital given orally through the day are helpful. Gray[4] recommended DILAUDID® or DEMEROL® administered hypodermically as being less apt to produce nausea than is morphine during the period of active bleeding.

Hypodermic injection of 0.6 mg. of atropine every six hours or intravenous injection of 50 mg. of BANTHINE® is often useful during the period of active bleeding to reduce motor and secretory activity of the stomach, to check gastric secretion and to eliminate antral spasm. Later, when the bleeding subsides, oral belladonna, atropine or banthine may be substituted. Since a vitamin C deficiency has been demonstrated in patients with bleeding ulcers, Gray[4] suggested the daily use of 100 mg. of vitamin C parenterally during active hemorrhage. This can be given orally when the bleeding has ceased.

Finally, although conservative medical management is the treatment of choice for massive hemorrhage from peptic ulcer, it is very wise to have the patient seen by a competent surgeon early in the course of such treatment. Those patients who continue to bleed in spite of adequate transfusions and the other therapeutic measures described, or whose hemorrhage ceases temporarily and then commences massively, should be given the advantage of emergency surgery before they lapse into irreversible shock. Aaron, et al.[1] have described a "test of transfusion" which is well worth following as a guide to the need for emergency surgery in patients with continuing massive hemorrhage. Also, some patients who respond well to conservative therapy may need elective surgery after the blood volume has been restored and all active bleeding has ceased.

Any patient who has recovered from a massive hemorrhage deserves a very careful follow-up study to determine whether he can safely be continued on a medical regimen or whether elective surgery should be advised.

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Treatment of Diabetic Coma

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SINCE 1923 the results obtained in the treatment of diabetic coma at the New England Deaconess Hospital have been reported at intervals.[1] During 1949 and 1950, 49 cases of diabetic coma have been treated at the New England Deaconess Hospital without a fatality. Recent emphasis on electrolyte balance and exchange, intracellular dehydration, the relation of glucose administration to insulin dosage and certain vascular complications have led to this evaluation and restatement of the methods employed.

J. W. STEPHENS

H. F. ROOT

Clinical Material

Our clinical material consisted of 789 patients seen from 1923 through 1950. During this period 2 series of patients were treated—the first consisting of 92 consecutive cases and the second of 49 cases—without a death. Admittedly these results were obtained because no patient appeared with such hopeless complications as carcinomatosis, fatal coronary or cerebral arterial occlusion or terminal vascular nephritis. Figure 1 shows the reduction in mortality that has occurred since 1923. From 1923 to 1939 the percentage of cases failing to recover was 11.9; from 1940 through 1948, 2.6 per cent failed to recover. This period included a series of 92 consecutive cases of diabetic coma treated without fatality. There has been no instance of fatality in a patient with either complicated or uncomplicated diabetic coma during the past two years.

Figure 2 shows that the average dose of insulin given during the first three hours of coma therapy following admission to the hospital has risen steadily from 83 units to 253 units. It is our conviction that the trend through the years toward earlier use of larger doses of insulin has been the major factor, not only in the successful treatment of severe acidosis but also in the earlier diagnosis and treatment of complications that may have contributed to the onset of acidosis.

The recent series of 49 patients included 32 women and 17 men. In this group 10 were recorded as totally unconscious on admission, 32 were drowsy but could be aroused, and 7 were only dyspneic. Data regarding these 49 cases are presented in Table 1. This information has been divided into 3 groups according to the admission blood sugar level. Shown in this table are the number of patients for each division, as well as the average age on admission, duration of diabetes mellitus, certain blood determinations and the amounts of insulin and fluid administered. It is note-

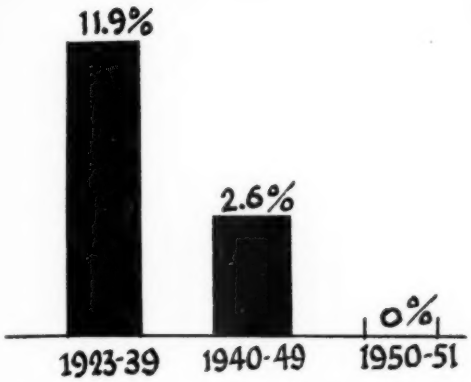

FIGURE 1. Reduction in case fatality in 789 cases of diabetic coma (blood carbon dioxide less than 9 mEq. per liter).

worthy that each increasing level in the blood sugar values was associated with an increase in duration of diabetes as well as a decrease in range of admission blood pressures. The lowest range of blood pressures is found in the group with the highest admission blood sugar values, probably the result of a more severe dehydration, a more marked ketosis and other unknown factors leading to deeper levels of unconsciousness, as well as shock.

This table also reveals that the dosage of insulin in the first three hours for patients with a blood sugar value over 1000 mg. per cent was more than 400 units, when it is noted that an average of 66 units was administered before admission. This is two and a half times the amount of insulin given in a similar period when the blood sugar was below 600 mg. per cent. Because of the extreme degree of dehydration associated with the higher blood sugar values (just over 2000 cc. of normal saline was required in the first group) greater amounts of parenteral fluid were administered—amounting to over 4000 cc. of normal saline in the third group—when the blood sugar on admission was 1000 mg. per cent or higher.

Physiopathology

Many of the metabolic and functional derangements accompanying severe diabetic acidosis are now more fully appreciated and have been recently reviewed by Guest[2] and Butler.[3] These disturbances are graphically demonstrated in Figure 3. From this diagram it will be seen that there are three major upsets which may be demonstrated by blood chemistry studies: (1) hyperglycemia, (2) metabolic acidosis and (3) dehydration. It will also be seen that hyperglycemia is the result of decreased glycogenesis, increased glycogenolysis and increased tissue catabolism.

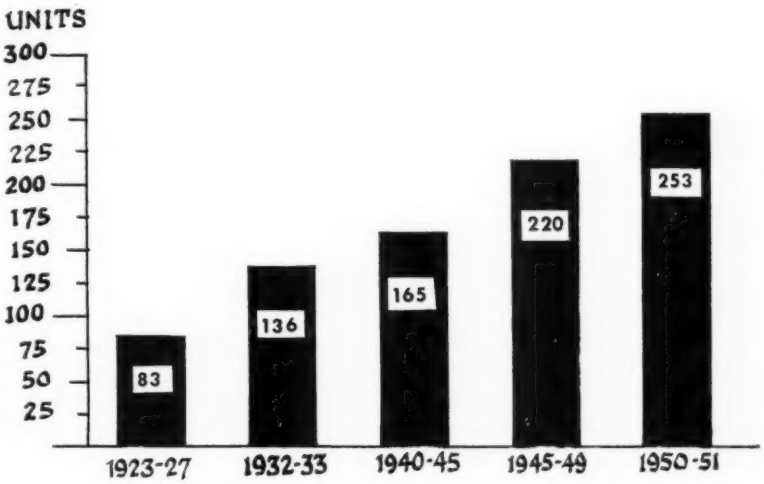

FIGURE 2. Increase in the average dose of insulin given to coma patients in the first three hours following admission to hospital.

TABLE 1

CONDITION AND TREATMENT IN RELATION TO ADMISSION BLOOD SUGAR VALUES

	BLOOD SUGAR VALUES		
	250-599 mg./100 ml.	600-999 mg./100 ml.	1000-1340 mg./100 ml.
Cases	24	19	6
Age	25	39	30
Duration of diabetes mellitus	8 years	9 years	13 years
Admission CO_2, mEq./liter	6.6	6.0	4.5
Admission nonprotein nitrogen, mg./100 ml.	44 average of 16 patients	75	66
Range of blood pressure	180/100-94/58	160/100-70/40	96/60-0
White blood cell count	14,845	21,752	17,140
Insulin prior to admission	52 units	39 units	66 units
Insulin first 3 hrs. after admission	148	321	455
Insulin first 24 hrs. after admission	229	475	705
Milliliters of intravenous normal saline	2340	3105	4050

When it is realized that prolonged anorexia, nausea and vomiting often precede the onset of severe diabetic acidosis, then it is appreciated that increased intracellular catabolism is probably the main contribution to the hyperglycemic state. This whole metabolic derangement is the result of insulin insufficiency or ineffectiveness indicated by increasing insulin resistance. This deficiency of insulin, permitting the appearance of hyperglycemia, also permits the other metabolic disturbances mentioned. Thus, as the liver becomes unable to form glycogen, it also becomes unable to metabolize ketone bodies, with the result that they begin to accumulate in the blood stream. Accompanying the increase in tissue breakdown is the release of mineral electrolytes, nitrogen, phosphoric acid esters and other acid metabolites which, along with the accumulation of ketone bodies, results in metabolic acidosis. A final disturbance, which is demonstrable both clinically and chemically, is dehydration. As the blood sugar rises, glucose excretion by the kidneys requires additional amounts of water. Thus, hyperglycemia stimulates glycosuria and polyuria with an accompanying loss of such electrolytes as sodium, potassium and chloride.

As dehydration becomes prominent, first the extracellular fluid compartment and eventually the intracellular fluid stores are depleted. Intracellular dehydration undoubtedly leads to other disturbances of which we know little, but they are possibly the most important mechanisms impaired as a result of diabetic coma. Thus, we would refer to the processes of tissue respiration and other enzymatic functions which are essential to the life of the cell.

From the chart we would expect then to find diabetic coma accompanied by such signs of dehydration as dry skin and dry tongue, hemoconcentration and decreased circulating blood volume. Other disturbances usually readily detected are the presence of glucose, diacetic acid and acetone in the urine, accompanied by a decrease in urine volume. As acidosis and dehydration advance impairment of renal function may result in the disappearance of glycosuria and ketonuria as death approaches. Examination of the blood exposes the presence of acetone bodies in increasing amounts, a decrease in the *p*H of the blood, an elevation in the blood pyruvic acid level, as well as some degree of hypernatremia,[4] and hypochloremia with the serum

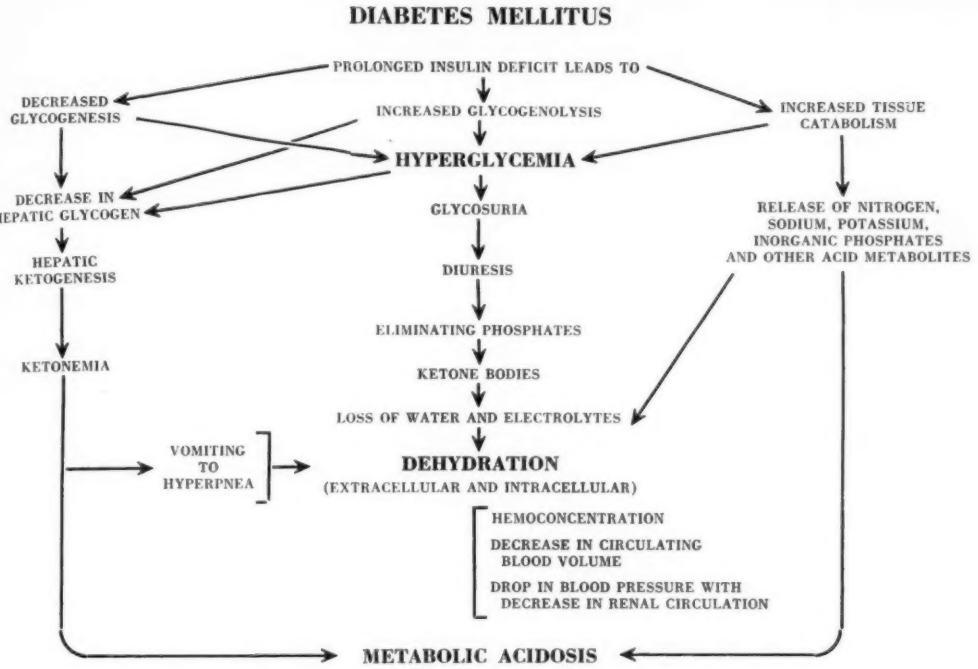

FIGURE 3

protein level undisturbed or slightly elevated. Increased tissue catabolism results in an increase in urinary nitrogen excretion, loss of intracellular potassium accompanied by continuous urinary loss of potassium, as well as phosphatemia and phosphaturia. Therefore, before treatment is begun for diabetic coma, the patient has had depletion of his glycogen, protein and fat stores in addition to loss of body electrolytes, water and other nutrients essential for normal tissue cell metabolism.

With rehydration, serum potassium levels usually fall as the urinary elimination of potassium usually resumes, and potassium again passes into the tissue cells during glycogen and protein storage. If glucose solutions are given parenterally early in the treatment of diabetic coma, the loss of potassium from the serum is enhanced and the amount of sodium and chloride lost in the urine is increased.[5] From this then, it must be realized that diabetic coma results in multiple electrolyte and other derangements that greatly affect the tissue cells, causing a disturbance of the normal blood chemistry, and all are the result of (1) insufficient insulin, (2) dehydration and (3) such consequential disturbances as metabolic acidosis, shock and impaired tissue cell metabolic processes.

Objectives of Treatment

From the foregoing presentation of the metabolic disturbance in diabetic coma, the following objectives of treatment may be offered:

1. Replacement of insulin deficiency. The primary and most urgent need in the treatment of diabetic coma is the early use of sufficient insulin. Between 1940 and 1951, 193 patients have had between 10 and 420 units of insulin administered prior to their admission to the hospital. This insulin in most cases was prescribed by telephone order before the patient left for the hospital. On the average these patients received 66 units of insulin before admission to the hospital and an

average of 165 to 253 units during the first three hours following admission. It now appears that routine orders of 20 to 50 units each half-hour are entirely out of place. Our main objective in the first three hours of treatment must be to note the admission blood sugar level, the amount of dehydration and the degree of disturbed consciousness and to administer sufficient insulin to effect the necessary extracellular and intracellular alterations to stimulate recovery.

2. Rehydration of the intracellular spaces.

3. Restoration of the electrolyte balance.

For these purposes parenteral fluid administration as well as the early use of food is desirable to furnish the necessary minerals, proteins and vitamins.

Treatment of Diabetic Coma

The most recent modification of the outline for treatment of diabetic coma at the New England Deaconess Hospital is shown in Table 2. Diabetic coma requires emergency procedures, the constant attendance of a trained nurse and constant medical supervision during the first few hours of treatment. A proper history and thorough physical examination are required immediately to ascertain the cause of coma, to appraise its severity and to discover complications. Immediately on admission urine is obtained—by catheter if necessary—for estimation of sugar, diacetic acid, albumin, casts and degree, if any, of pyuria present. A blood sample is also immediately drawn for determination of the sugar and carbon dioxide content, blood acetone bodies, nonprotein nitrogen value and a white blood cell count. Other laboratory procedures of value are the serum potassium evaluation, electrocardiogram and x-rays of the chest and abdomen if indicated. These procedures help to evaluate the depth of coma as well as to locate complications when present.

Insulin should be administered immediately on admission, 100 units of regular insulin being given subcutaneously for adults and even 200 to 300 units may be given, in part intravenously, if the patient is in shock and unconscious. On receipt of the admission blood sugar value, a further dose of insulin is given equivalent to an additional 200 units if the level is over 600 mg. per 100 cc. and an additional 300 units if over 1000 mg. per 100 cc., especially if the patient is unconscious. Normal saline is used to combat dehydration and at least 2000 cc. is administered initially by drip.

The extent of dehydration and the amount of fluid to be given are difficult to estimate. Clinically, maximum dehydration is accompanied by anuria which is always a terminal stage in diabetic coma unless death comes from some other complication. When anuria is present, due solely to diabetic coma and not to some complicating renal or cardiac lesion, the administration of fluid may be at the rate of 1000 cc. per hour intravenously. If the patient, with a catheter in the bladder, is excreting urine at the rate of 10 to 20 cc. per hour, severe dehydration may be assumed. If, however, the rate is from 25 to 50 cc. per hour, the dehydration may be severe but certainly is not critical. If, as the result of shock, the blood pressure has been greatly lowered and anuria appears to persist, the possibility of lower nephron nephrosis must be borne in mind. In such patients excessive fluid administration—beyond approximately 4000 cc. in 24 hours—is to be avoided.

When lower nephron nephrosis is suspected, 10 per cent glucose may be administered intravenously in amounts to compensate for fluid loss by sweating, respiration, vomiting, etc. If there is no vomiting, about 1500 cc. of 10 per cent solution is permitted each day. This amount is increased by the amount lost by any other route. The use of insulin together with the glucose solution may prevent a dangerous rise in blood potassium values. It is well to eliminate the oral route for fluid administration as this often precipitates nausea and vomiting. Usually recovery occurs, sometime after six to nine days.

It is also well to recall that patients with signs and symptoms of hypokalemia, as well as those who have died, have been those who have received glucose solutions intravenously. Thus, in the early hours of managing the patient in diabetic coma one must refrain

TABLE 2

Treatment of Diabetic Coma

FIRST HOUR AFTER ADMISSION

Assign special nurse, preferably experienced in coma treatment, for the first few hours.

LABORATORY

1. *Urine*—Examine for sugar, diacetic acid, albumin, coma casts and pyuria. Catheterize if necessary.
2. *Blood*—Test for sugar and carbon dioxide content, with emergency report inside the hour. White blood cell count and nonprotein nitrogen also of value. Qualitative acetone desirable, serum potassium helpful.
3. *Search for complications.*
   - A. History to explain cause of coma.
   - B. Physical examination, noting particularly (a) state of consciousness, type of respiration, pulse rate, blood pressure and rectal temperature, and (b) look for soft eyeballs, dry tongue, dilated stomach, cold and mottled skin and impacted rectum.
   - C. X-ray chest and abdomen when possible.
   - D. Electrocardiogram—(a) coronary and (b) potassium changes.
4. *Insulin*—100 units of regular insulin subcutaneously at once for adults, if blood sugar exceeds 300 mg. per hundred cubic centimeters and if the blood carbon dioxide content is 9 mEq. per liter (20 volumes per cent) or less. The dose would be proportionately less in cases of recent onset of diabetes or in young children. In cases with blood sugar between 600 and 1000 mg., give 200 units additional, and with blood sugar over 1000 mg., give 300 units additional. In patients in circulatory collapse, give preliminary additional doses of insulin intravenously.
5. *Gastric lavage*—Use large tube, aspirate completely and wash stomach with warm water with greatest care.
6. Normal saline intravenously, 2000 cc. and repeat if indicated by dehydration and blood pressure below 90 mm. of mercury. Avoid too rapid administration, especially in older patients.
7. Keep patient warm yet avoid burns, as from hot water bottle.
8. Give no potassium solutions by mouth or by vein (a) when oliguria is present or impending, (b) unless blood analysis or electrocardiogram clearly indicates hypokalemia, and (c) as a rule until after 12 hours of treatment.

SECOND TO SIXTH HOUR

The gravity of the condition of the patient may require repetition of first hour's total insulin in the second hour.

9. Repeat blood sugar and carbon dioxide determinations after three hours. For rising blood sugar give insulin hourly 50 to 200 units or more, according to physician's judgment of prognosis.
10. *Fluids by mouth*—As soon as tolerated, give 100 to 120 cc. per hour of broth, ginger ale, orange juice, tea or coffee, to be sipped by patient or spooned by nurse. If nausea and vomiting recur, withhold fluids orally for 12 hours (lavage stomach again if indicated) and then resume.
11. *Enema* for cleansing and to relieve abdominal distention.
12. Record and note changes in blood pressure, pulse and temperature hourly. Consider transfusion if in deep shock.
13. Urinalysis for sugar and diacetic acid every hour. Record hourly output as index of dehydration.
14. Antibiotics (parenteral)—Penicillin and streptomycin frequently needed.

SIXTH TO TWENTY-FOURTH HOUR

15. Repeat blood sugar and carbon dioxide determinations. Give insulin 50 to 200 units if blood sugar and carbon dioxide levels are not improving. Insulin (regular) may be given according to urine tests every one to four hours if fall in blood sugar has been satisfactory.

If test is—Red	Orange	Yellow	Green	Blue	
Give— 20	16	12	0	0	units

16. Soft or liquid food such as oatmeal gruel, orange juice or milk diluted half and half with lime water, not to exceed 10 gm. carbohydrate per hour. Glucose (5 per cent in saline) intravenously only when blood sugar approaches normal.
17. *Urinary output*—Observe this closely and note with alarm any sign of anuria. Treat with 1500 cc. intravenous saline if shock is persisting. Repeat as necessary. For anuria associated with hypochloremia give 50 cc. of 10 per cent salt solution intravenously. Never give hypertonic glucose solution to promote diuresis. Beware of producing excessive diuresis with consequent loss of base, especially of potassium.
18. Sudden onset of muscular weakness and shallow respiration suggest hypokalemia. Potassium may be given by mouth or intravenously if changes in electrocardiogram or in serum potassium are present, rarely exceeding a total of 3 gm. potassium chloride by mouth in three to six hours.

SECOND DAY

19. Soft food—Diet: carbohydrate 100 to 150 gm., protein 50 gm. and fat 50 gm.
20. Protamine zinc or NPH insulin should be begun, supplemented by regular or crystalline insulin in small to moderate doses before meals, as indicated by blood sugar and urine tests.

THIRD DAY

21. Patient should gradually return to the standard diabetic diet for age and weight with carbohydrate 150 to 200 gm., protein 60 to 100 gm. and fat 60 to 120 gm. daily.

TABLE 3

ADMISSION AND TREATMENT DATA FOR 6 PATIENTS GIVEN COCARBOXYLASE

CASE	SEX	AGE AT COMA	DURATION DIABETES MELLITUS	DEGREE OF CONSCIOUSNESS	BLOOD SUGAR *(mg./ 100 ml.)*	CO_2 *(mEq./ liter)*	N.P.N. *(mg./ 100 ml.)*	INSULIN FIRST 3 HRS.	INSULIN FIRST 24 HRS.
1	M	39	2.0	D	413	7		2240	7810
2	F	22	5.0	U-3	400	6	34	280	320
3	F	67	3.0	D	980	4		300	461
4	F	14	12	D	390	9	38	100	232
5	M	30	16	D	1340	8	83	400	1206
6	F	17	7	N	635	7		350	492

from using intravenous glucose if one hopes to prevent hypopotassemia. It is well also not to use such solutions because of their dehydrating effects as has been pointed out by Lee, Naidoo and Toriens.[6] They compared the therapeutic results in 2 series of patients receiving glucose solutions with those of others treated without the use of glucose. Mortality in the first series was four times as great as that in the second.

Recently a series of 17 patients was studied by Seldin and Tarail[5] who used massive doses of glucose solution in one group of patients and only moderate amounts in another group. Their tables show an augmentation of water and salt excretion with massive injections of glucose, explained by the fact that salt as well as water is swept into the urine with glucose. Such injections of glucose, in stimulating a marked diuresis, caused a loss in urinary glucose equal to 50 per cent of the glucose administered. In 1 patient given 50 cc. of 5 per cent glucose and an infusion of 750 cc. of 10 per cent glucose the serum concentration of sodium and chloride fell from 116.9 to 87.5 and from 91.6 to 71.1 mEq. per liter respectively. The fall in serum chloride values was clearly due to a movement of water out of the cells in response to the change in osmotic pressure resulting from glucose. Urinary loss of salt played no part, as the patient was anuric. Results of this cellular dehydration are probably most disturbing although their effects are not clear.

In our series of patients the fluid administered has been predominantly physiologic salt solution. Whether this or the fact that many of our patients receive insulin early, prescribed by telephone before leaving for the hospital, has been a factor in reducing the frequency of low serum potassium, anuria and other complicating factors is hard to determine. The simplicity of administering physiologic salt solution for the doctor who sees patients at home or who does not have laboratory services has seemed, in view of our lack of case fatalities, to justify a continuance of its use. The average coma patient receives 2000 to 3000 cc. intravenously in the first three to five hours of treatment. In severely dehydrated cases as much as 8000 to 10,000 cc. has been given.[7]

Transfusions have been employed in only 3 cases. In one a bleeding duodenal ulcer complicating diabetic coma was a definite indication. In the others a severe drop in blood pressure was the indication. We feel that the primary disturbance accompanying the appearance of shock in diabetic coma is the marked decrease of intracellular dehydration present. Fluids, such as plasma and blood, which remain in the blood stream do not really aid this intracellular disturbance. In fact, in older patients where coronary disease and myocardial infarction may be lurking in the background, excessive expansion of the circulating blood volume by fluid not transferable to the intracellular confines may precipitate cardiac decompensation.

One objective in the first few hours of treat-

TABLE 3 (continued)

W.B.C.	B.P.	BLOOD ACETONE *(mg./100 ml.)*	SALINE (ML.)	COCARBOXYLASE
				Insulin allergy and insulin resistance
11,900	104/50		2500	Cocarboxylase 50 mg. I.V. repeated in 2 hrs.
12,800	108/70	104	3000+	Cocarboxylase 100 mg. I.V. repeated in 1 hr.
15,750	168/88	37	3000	Cocarboxylase 100 mg. I.V. every 2 hrs. for 3 doses
18,300	118/80	95	2000	Cocarboxylase 100 mg. I.V. every 2 hrs. for 2 doses
11,450	96/60	102	6000	Cocarboxylase 100 mg. I.V. every hr. for 4 doses
12,200	120/70	137	3000	Cocarboxylase 100 mg. I.V. every hr. for 2 doses

ment is to clear the stomach by gastric lavage, relieving gastric dilatation and the toxic effects of the gastric contents. This makes possible the early ingestion of food. In many patients foods, including orange juice, oatmeal and chicken broth, can be offered after six to eight hours. However, in those patients who remain unconscious or are unable to take food by mouth for periods of 24 to 48 hours, nutrition must be provided intravenously. If renal function is adequate and the blood chemistry remains normal multiple electrolyte solutions, such as the one recommended by Butler[3] which contains glucose, sodium chloride, phosphates and lactates and such electrolytes as sodium, potassium and chloride, become of great value. To this solution it is well to add multiple B vitamins and vitamin C.

In the past, caffeine, sodium benzoate and other drugs have been employed. At present practically no drugs are used, except occasional doses of caffeine or adrenocortical extract.

Enzyme disturbances may play a more important part in diabetic coma than has yet been demonstrated or comprehended. Professor Boulin[10] described the use of cocarboxylase in diabetic coma. He attributed the success of this substance, which is also known as thiamine pyrophosphate, to its role in pyruvic acid conversion. It is to be noted that his patients were given large amounts of glucose and the amount of insulin required was far in excess of that required in the average case treated in this clinic. However, in an attempt to investigate these questions, a series of 6 cases shown in Table 3 were given cocarboxylase at the Deaconess Hospital in 1949. It will be seen that in 1 case of this group insulin resistance was already present and the patient went into diabetic coma while taking 1000 units of insulin a day. He required 8000 units for recovery from diabetic coma. He received 50 mg. of cocarboxylase every two hours. In the remaining patients of this series cocarboxylase was injected intravenously in doses up to 100 mg. every hour for four to five hours. It will be seen from Table 3 that the amount of insulin required by these patients does not differ from that used in cases averaged in Table 1 and in this small series we were unable to demonstrate any specific value of cocarboxylase.

Complications of Diabetic Coma

Certain remediable or preventable complications associated with diabetic coma are to be borne in mind. Thus, aspiration pneumonia may result from failure to wash out the distended stomach. Electrocardiographic studies may disclose otherwise silent or unsuspected myocardial infarction. Hypokalemia, which appears during the recovery stages of diabetic coma, has attracted much attention since Holler's observations.[8] This metabolic upset has been recently commented on by Stephens.[9] Among 181 admissions for diabetic coma to the Montreal General Hospital, 4 patients showed clinical evidences of disturbed potassium balance. The patients who

TABLE 4

METABOLIC RATE DURING DIABETIC COMA

CASE	SEX	AGE AT COMA	WEIGHT (POUNDS)	HEIGHT (INCHES)	DATE	TIME	O_2/MIN.	CAL./24°	B.M.R.	B.S., MG./100 CC.	CO_2, MEQ.	COMPLICATIONS
1	M	29	127.6	73	11/8/35	4:45 P.M.	286	1987	+16	1070	4	Varicella
						9:45 P.M.	385	2675	+56			
					11/9/35	8:00 A.M.	317	2202	+29	110		
2	F	39	112.2	62.5	5/27/36	4:45 P.M.	283	1965	+50	410	5	
					5/28/36	8:00 A.M.	206	1434	+11	040	10	
3	M	21	136.4	69.5	5/31/36	12:40 P.M.	321	2234	+26	620	1	Hepatitis
					6/2/36	8:00 A.M.	334	2318	+30	70	20	Temp. 103° F.
4	F	37	101.2	61.5	3/2/37	2:03 P.M.	191	1327	+11	810	3	
					3/3/37	8:00 A.M.		1529	+25	160	7	
					3/5/37	8:00 A.M.		1355	+11	37		
5	F	32	118.8	68.5	11/3/35	1:40 P.M.	260	1806	+27	600	5	
						5:40 P.M.	223	1549	+10	470	7+	
					11/4/35	8:00 A.M.	270	1529	+ 7	50	9	
6	M	47	130.0	60.0	11/21/40	8:55 A.M.	249	1730	+23	490	14	
					11/23/40	8:35 A.M.	225	1563	+11	320	19	
					11/26/40	9:00 A.M.	228	1528	+ 8	270		

developed flaccid paralysis and respiratory difficulty in association with hypokalemia were those who had received intravenous glucose. When large doses of insulin are used together with glucose solution, the rate of decline in serum potassium is accelerated. Severe insulin resistance, sometimes secondary to fever or hyperthyroidism, may occur. Less commonly, actual insulin insensitivity may lead to severe diabetic acidosis. In such cases it is probably well to give insulin intravenously to promote more rapid action and also to prevent the formation of insulin depots. Infection of the genitourinary tract during the use of the urethral catheter is a major hazard, and the use of antibiotics is a preventive measure. Most important is the hourly test of the urine and repetition, every three to six hours, of the blood sugar test in order to recognize, prevent or treat serious hypoglycemia. Large doses of regular insulin have a prolonged action. Adequate fluid administration will prevent critical dehydration, shock and anuria.

Arterial thromboses in the legs, brain or heart occurred during the dehydrated stage of coma in 5 patients during the last four years. Thrombosis probably occurred because of the changes produced by dehydration and reduced blood volume in arteriosclerotic tissues.

During diabetic coma a marked increase in the respiratory rate and volume is observed and a correspondingly high oxygen consumption is noted. This high metabolic rate is sometimes difficult to interpret because of the presence of complications which

may not be easily recognized in the comatose patient. A high metabolic rate induced by muscular exercise usually lowers the blood sugar provided an adequate supply of insulin is present. On the other hand, in diabetic acidosis when the metabolic rate is high the rapidity of development of acidosis is increased. Thus, in the case of Mrs. M. diabetic coma developed in a few hours and the patient's metabolism was determined by the same method used in a series of patients reported in Table 4. This method was devised by Professor Francis G. Benedict at the Carnegie Nutrition Laboratory. Measurement of the oxygen consumption of a patient in bed in the hospital ward was possible by using a bucket with a tight fitting rubber diaphragm around its neck. In the patients described in Table 4 who came into the hospital unconscious, after many hours of vomiting, the metabolism was determined at the hour of arrival. This was not a basal test except that it could be said that these patients really had been fasting for some hours. It is seen in the table that high metabolic rates were the rule during the first few hours of coma. Complications noted in these patients included varicella, hepatitis with a temperature of 103° F. in Case 3, and primary hyperthyroidism in the case of Mrs. M. whose metabolism was +70 per cent. Following recovery from coma with the usual treatment, she was operated on and a subtotal thyroidectomy was carried out for primary hyperthyroidism.

In hyperthyroidism without diabetes the amount of glucose which can be burned by the patient is exceptionally high. Observations were carried out in a few hyperthyroid patients by the administration of glucose solution intravenously and it was clear from the high and continued respiratory quotients that these patients are able to utilize glucose at a high rate. This was shown many years ago by Wilder and Woodyatt when they gave continuous intravenous administration of glucose solution and determined the rate of glucose utilization per hour per kilogram of body weight before glycosuria occurred. On the other hand when the insulin supply is limited or its action impaired, as in true diabetic coma, the high metabolic rate adds materially to the rate of ketone formation and protein breakdown with consequent glucose formation from protein and therefore itself is a contributory factor influencing the rate of development of ketosis and its severity.

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DIAGNOSIS BY INTUITION

DIAGNOSIS by intuition is a rapid method of reaching a wrong conclusion. . . . *John Chalmers Da Costa, Selected Papers and Speeches.*

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ARMED FORCES INSTITUTE OF PATHOLOGY

CONTINUED interest in the Armed Forces Institute of Pathology by American medical, dental and veterinary societies, along with the enthusiastic support of a large number of civilian pathologists throughout the United States, has added new and additional responsibilities to AFIP which make it of even greater significance to military and civilian medicine than ever before.

One of the most significant features of AFIP which sets it apart from all other institutions in the world today is the vast collection of well documented medical and surgical cases, together with the related pathologic specimens and illustrative material of servicemen in the military age group of 18 to 38 years. Because of these facts, in a special field where service is by far the most important link in any lifeline, the AFIP in Washington, D.C., occupies a unique position in relation to "help wanted" calls from armed forces medical units and members of the civilian medical profession located all over the globe. This material has been accumulated over a period of more than 90 years during which there were four wars and a long Indian campaign. An understanding of the present organization and operative procedures of this institution, and the utilization of the pathologic material which is continuously being assembled, is of great importance in conjunction with the role it now plays in pathology throughout the United States, and the potentialities it may have for greater usefulness in this field in the future.

Four major departments, each of national import, comprise the framework of AFIP—the Department of Pathology, the American Registry of Pathology, the Medical Illustration Service, and the Medical Museum. All are jointly staffed and equally shared by the Army, Navy and Air Force.

The Department of Pathology is one of the Institute's most highly diversified and specialized activities. Day after day, surgical and autopsy materials from widespread sources are received in the departmental laboratories. In the course of a typical day, the professional staff processes more than 135 cases for diagnosis, consultation and review. The facilities of AFIP are used by the medical services of the Army, Navy, Air Force, Veterans Administration, U.S. Public Health Service and other federal agencies.

A very important part of the Institute's program is the American Registry of Pathology, an administrative facility, which serves as a central agency for the collection of pathologic material, pertinent clinical records and laboratory data in specific fields of pathology. It is composed of 20 individual registries which are sponsored by the various nation-

al medical, dental and veterinary societies.

Each registry specializes in the diseases of its particular field, such as the eyes, skin, oral regions, bone, and other organs and body systems. The registries are of extremely great value to civilian practitioners who may submit their pathologic material for consultation through their local pathologists. Thus, the Institute in no way competes with civilian pathologists but, rather, strives to be of help to them in a consultative capacity.

Among the earliest functions of the Institute was the Photography Department which, in the postwar period, has expanded to become the Medical Illustration Service. This service was recently reorganized and integrated with the training film activities of the Surgeon General. Under its jurisdiction come all aspects of medical art, design and illustration handled by the Institute. The Illustration Service is also responsible for the development, construction and maintenance of all Medical Service exhibits including those which are installed in the Medical Museum. These exhibits are available to all professional medical societies. Supervision of clinical photographic activities in all armed forces hospitals is another responsibility of the Medical Illustration Service. For example, a central file of illustrative material is maintained which is available to all medical personnel of the federal services, medical schools, medical organizations and civilian practitioners.

Arrangements are currently being made for the Medical Museum ultimately to provide the civilian medical profession with comprehensive study exhibits illustrating all phases of disease. These will include history, diagnosis, pathology, etiology, prevention and treatment of tumors and other diseases. Exhibits for the public are prepared as educational activities, with displays constantly being changed to conform with changing interests.

Medical services of the Army, Navy, Air Force, Veterans Administration, U.S. Public Health Service and civilian medical agencies throughout the world send surgical and autopsy data to the AFIP departmental laboratories. All materials and records received are indexed as to origin, diagnosis and contributor, thereby constituting an invaluable source for research and statistical analysis.

Another of the Institute's more important roles, of which it is quite proud, is the part it plays in providing specialized training for qualified medical, dental and veterinary personnel. It is accredited for one year advanced training in preparation for the American Boards of Pathology, Dermatology, Syphilology and Oral Pathology. Procedures have been initiated to secure similar credit from boards of other medical specialties. The AFIP has prepared numerous sets of microscopic slides and related texts which may be borrowed by students who cannot conveniently come to Washington. These may be procured on loan through the American Registry of Pathology and cover practically every field of medicine.

Training within the Institute is conducted in a variety of ways. Medical officers of the Army, Navy, Air Force, Veterans Administration, U.S. Public Health Service and other federal agencies may apply for this training. Officers of the Medical and Dental Reserve Corps may also be ordered to active duty for such training purposes. Research and training fellowships for civilians have been established by several medical schools, the American Dental Association, and the American Veterinary Medical Association.

The Armed Forces Institute of Pathology today represents an unlimited horizon for the study of diseases and tumors and their correlation with clinical observations. Unique in its field, AFIP has become a national institution for the diagnosis, collection and preservation of specimens and records relating to all diseases. With the advent of its new building, currently being constructed on the grounds of the Walter Reed Army Medical Center, greatly expanded research facilities will make it possible to increase tremendously the Institute's services to military and civilian medicine alike.

GEORGE E. ARMSTRONG, MAJOR GENERAL
The Surgeon General
Department of the Army

MEDICAL EXAMINERS OR THE CORONER SYSTEM

In 1855 a committee was appointed by the House of Delegates of the American Medical Association to report what measures should be adopted to remedy the evils then existing in the methods of holding coroner's inquests "by which the lives and liberties of the innocent may be jeopardized, and the ends of justice frustrated." The report of the committee in 1857 indicated the need for reorganization of the coroner's office. Nothing much happened but by 1887 reforms were proposed including:

1. Abolition of the office of coroner.
2. Dispensing with jury services in the coroner's office.
3. Separation of medical from legal duties in all cases where crime is suspected.
4. Entrusting medical examination only to competent officers.
5. Limiting medical officers to smallest number possible.
6. Consigning all questions of law to competent legal magistrates.
7. Removing politics from these appointments.

By 1910 a resolution called attention to the improvement that had been brought about in Massachusetts but the House of Delegates referred it to the committee on legislation. By 1911 the Report of the Council on Health and Public Instruction found out the problem was complicated. In 1941 Dr. Walter G. Phippen of Massachusetts introduced a resolution which pointed proudly to the Massachusetts record and the reference committee passed it over to the Board of Trustees. By 1943 studies were being made and in 1944 it was overlooked but in 1945 the Report of the Committee to Study the Relationship of Medicine to Law was approved by the Reference Committee on Legislation and Public Relations and adopted by the House of Delegates. Then in 1948 the Report of the Bureau of Legal Medicine to the Board of Trustees was referred to the Reference Committee and here is the progress:

"The Medical Examiner: It is noted with considerable interest that the coroner-medical examiner system is gradually replacing the old coroner system. Particular attention is called to the accomplishments in Virginia, while the medical examiner laws have also been improved in Maine, New Hampshire and New Jersey. Your committee wishes to reemphasize the previous pronouncements of this House in favor of the medical examiner system to replace the antiquated coroner system."

In an address before the American Academy of Forensic Sciences in 1951 Dr. Louis J. Regan, Los Angeles, pointed out the legal authorizations that are required for autopsy. Usually the coroner is vested with the power to perform an autopsy or to procure an autopsy. In a number of states the functions of the coroner are carried out by a justice of the peace; in still others, by such other agencies as the office of the medical examiner. In a single state (Oklahoma) there is a statute providing for postmortem cesarean section. Actually Dr. Regan indicates that Connecticut, Maryland, Massachusetts, New Jersey, New York, Virginia and Wisconsin provide for a medical examiner. Some states provide for a coroner, others for a justice of the peace and still others for a variety of appointees. The situation is confused and certainly one in which necessary legal bodies should be properly constituted by law to make the necessary decisions.

A special committee including Drs. Alan R. Moritz, Edward R. Cunniffe and Harris S. Martland, and Mr. J. W. Holloway, Jr., called attention to the following basic conclusions:

1. That, throughout most of the country, medicine participates less effectively in the administration of justice in the United States than in any comparable country in the world.
2. That the ineffectual manner in which medical knowledge and skill are utilized in the administration of justice in many jurisdictions of the United States undoubtedly predisposes to:
   a. The nonrecognition of murder.
   b. Unjust accusation of innocent persons.
   c. The improper evaluation of medical evidence bearing on the circumstances in which fatal injuries were incurred.
   d. Failure to acquire medical evidence which would be useful in the apprehension of criminals.

e. Failure to acquire medical evidence essential to the administration of civil justice.
f. Ignorance of certain otherwise preventable hazards to public health.
g. The impairment of the value of vital statistics.

3. That state and county medical societies should examine the laws that relate to the office of coroner or medical examiner in their respective communities with a view to securing such remedial legislation as may be desirable.

4. That consultation facilities should be made available through the Bureau of Legal Medicine and Legislation whereby the authorized representative of any state or county medical society may secure advice regarding the kind of remedial legislation pertaining to the office of coroner or medical examiner that would best serve the needs of the community in question.

Apparently at least five or six states have already decided the mechanisms by which legally constituted authorities will take the responsibility for determining the causes of death. Certainly it would seem to be simple for the experts to determine the causes of death and for the legally constituted authorities to apply the information that is here found to the necessary agencies for bringing to the courts the evidence necessary to convict. It seems strange that while most of the rest of the civilized world has moved forward, we here in the United States fail to make the necessary progress.

MORRIS FISHBEIN

CAREERS IN CARING FOR THE HANDICAPPED

ACCORDING to the National Society for Crippled Children and Adults, more than 115,000 opportunities for professional work with handicapped children and adults now await applications by those who have been suitably trained. Vacancies call for 4000 occupational therapists, 16,000 physical therapists, 12,000 to 15,000 speech therapists and some 80,000 specially trained teachers who can educate physically handicapped children.

The National Society for Crippled Children and Adults has cooperated with the National Foundation for Infantile Paralysis and with the Office of Vocational Rehabilitation of the Federal Security Agency in the preparation of a pamphlet entitled "Careers in Service to the Handicapped" which has been sent to the public and parochial school systems throughout the country. Copies are available through the Office of the National Society for Crippled Children and Adults at 50 cents each. The booklet provides full information as to training in the fields concerned and as to opportunities for employment.

MORRIS FISHBEIN

SELF-EXAMINATION OF THE BREAST

SINCE 1949 American physicians have been earnestly concerned with the proposal for teaching to women a simple technic for self-examination of the breast with a view to detecting cancer of the breast at the earliest possible moment. A motion picture for such education has been prepared and has been approved by the American Medical Association, the Public Health Cancer Association and similar groups.

In November 1949 female employees of the state government in Springfield, Illinois, were notified that a course of instruction in self-examination of the breast would be offered and women over 30 years of age were particularly encouraged to participate. Dr. G. Howard Gowen, deputy director of the Illinois Department of Public Health, now reports that 470 requests were received for such instruction and that about 80 per cent of the requests were from women 30 years of age and over. Less than one-third of the participants had a record of cancer in the family. In the course of instruction, the basic technic recommended by Dr. C. D. Haagensen, Presbyterian Hospital, New York City, was adopted. The instruction required about 10 minutes for each of the participants.

Two years later questionnaires were sent to the 470 women to whom personal instruction in self-examination of the breast had been given. One hundred and twenty-nine replies were received or a 27 per cent return. One hundred and two of the women were continuing the practice of self-examination, 9 had discovered tumors and in 1 instance the tumor was malignant. Sixty-four of the women had taught self-examination to others. Although the women had been told that examination less than once every two months was not needed, 40 of the women arbitrarily adopted a monthly interval between examinations.

Although only 129 replies were received to the questionnaires that were sent to 470 women, there is evidence that self-examination can be taught with advantage to women over 25 years of age and that it can serve as a means to finding a significant number of undiscovered tumors of the breast or even of malignant growth. In some states the film has now been shown to at least one-third of all the women in the state and new evidence will become available with time as to whether or not the use of the film alone or of personal teaching alone or of combined methods is most effective in securing useful results.

MORRIS FISHBEIN

MEDICAL SCIENCE IN GREAT BRITAIN

ONE hears nowadays so many different points of view regarding what is happening in Great Britain and these come from so many different sources that special interest naturally attaches to a comment made by Dr. E. D. Adrian[1] who is president of the Royal Society of London and who is at the same time one of the world's greatest investigators in the field of medical science.

Among the most significant of the statements made by Dr. Adrian was the following: "As you know, the inhabitants of Great Britain today are not so very far from being entirely dependent on the state. Most of our universities are maintained by state funds and nearly all the doctors and scientists get their income directly or indirectly that way. I don't think this is likely to change much in the near future whatever government we may get, but, so far, I don't think we have really suffered much from it. The University Grants Committee has been going long enough to have established the tradition that the universities must be allowed to run themselves. And as far as medicine is concerned, the universities control the teaching, so that there is not much danger that medicine will be forced into a set pattern unless it is just what the doctor orders.

"That, of course, is the danger that we have to face when the State takes charge of us—the danger that we shall all be forced into a set pattern. You don't need to be told that, for you have shown us all how much faster things can go under private enterprise. And private enterprise is the life blood of science: freedom to go off in new directions, to follow the unexpected observation, to make mistakes and waste one's time."

Dr. Adrian fears particularly the possibility that international organizations would now become so important that they might even dominate the trend of science more greatly than occurs in individual nations. He said, for instance, "There is even a danger nowadays that some lines of scientific work may become concentrated in institutes which are not merely national but are international. Here science would be without competition and it would make no mistakes, because there would be no one outside to show that we had made them. It would be science without tears, but without much chance of discoveries."

Already those who observe the trend toward international organization recognize the dangers that lie in bigness on either the national or international level. Mass action is usually unthinking action. Progress depends on freedom of the individual to think and work and the most that the mass can do is to encourage and aid the intelligent individual.

MORRIS FISHBEIN

1. ADRIAN, E. D.: Freedom and responsibility of science. News Report, National Academy of Sciences 11:49 (July-August) 1952.

MEN OF *Medicine*

He Made a Science of Child Development

DR. Arnold Gesell of New Haven, Connecticut, will go down in medical history as the individual who rescued "child development" from the limbo of empty abstractions. The results of his more than 35 years of continuous research have enabled physicians to evaluate accurately the developmental patterns of both normal and handicapped children.

Thirty-five years ago the welfare of the preschool child was a no man's land. Physicians called to see sick babies when summoned. Well children rarely came under trained observation and busy practitioners gave them scant heed. If the family physician suspected that a child was not developing normally, there was little constructive interpretation he could offer the parents. Often he would hesitate to voice suspicions because he lacked criteria for normal, and thus also for abnormal, development.

Only the more obvious cases of retardation aroused suspicions. Parents blundered along as best they could and ascribed the conduct of their offspring to inherent goodness or badness rather than to normal traits or deviations.

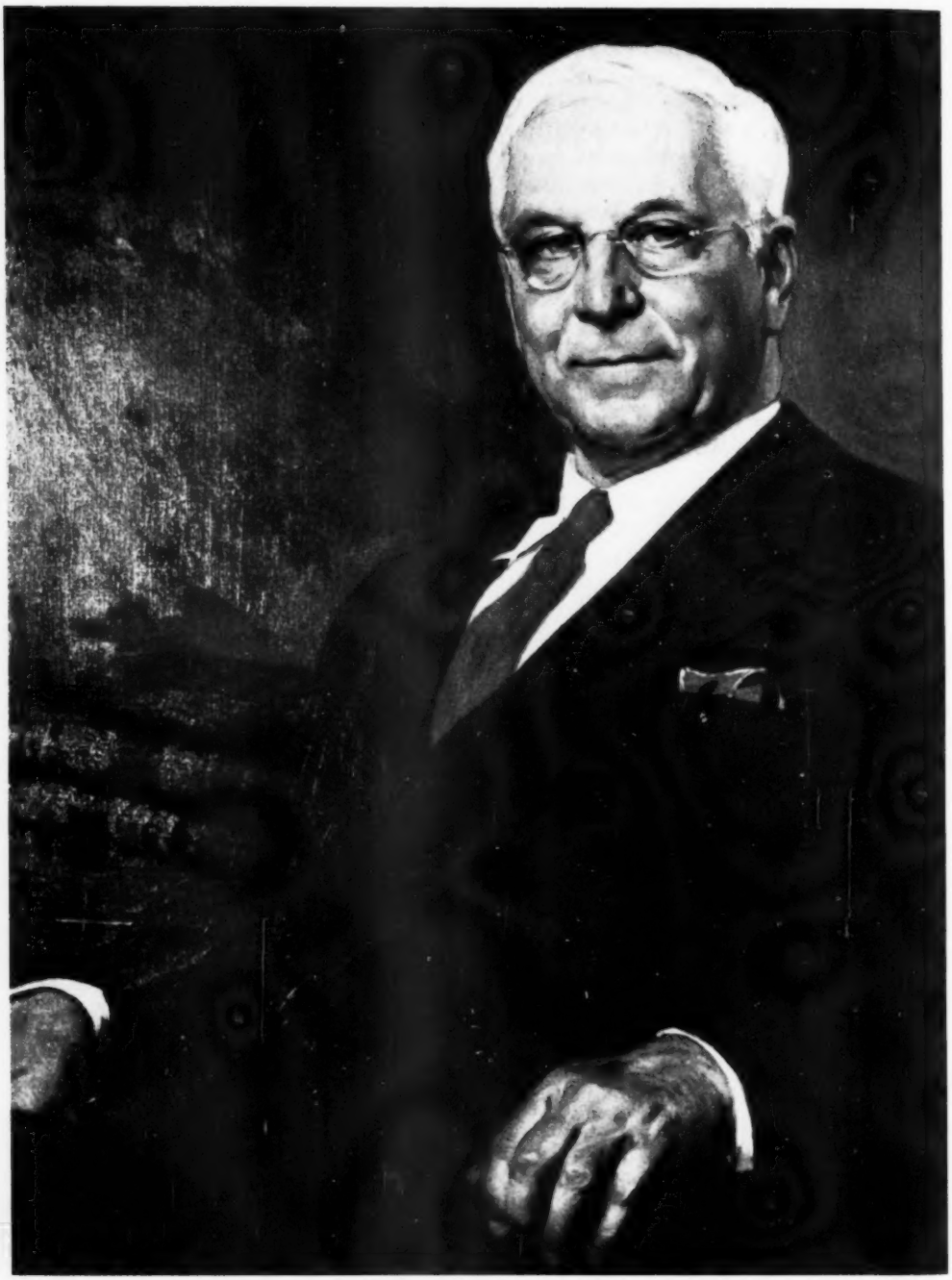
It remained for Dr. Gesell to produce a diagnostic method whereby normality could be appraised with the aid of norms of behavior development. But this method was not planned with any intention of pinning I.Q.'s on babies. The behavior tests were designed for the wholly constructive purpose of defining potentials of both normal and handicapped children and aiding them to reach their greatest possible development. Such developmental diagnosis serves to protect children against any hammer and tongs treatment.

Dr. Gesell says that he does not know when he started on the career which was destined to make one of the truly important contributions of the century. But certainly the Mark Twainish scenes of Gesell's childhood helped to foster an early and deep awareness of life and a profound understanding of people. Arnold Gesell was born in 1880 in Alma, Wisconsin, a picturesque, two street village on the banks of the upper Mississippi.

There as a boy he found plenty of opportunities for nonacademic as well as precurricular and extracurricular activities. He enjoyed a richness of firsthand experiences denied present day children who much too often snatch their adventures vicariously from radio, television, motion picture and comic book. Being personal instead of synthetic adventures, the experiences of his childhood were vivid and colorful.

There was the jubilant excitement of two long and three short blasts from the packet boat followed by the thrilling shout, "She's going to land," that passed from boy to boy. There was the rush to the dock to behold strange personages and happenings. There were the antics of Negro roustabouts from the land of cotton far down the river and the sight of brawny rivermen from nearby logging works.

Each holiday was special and meaningful, regaling the insatiable, tireless senses of a small boy. Memorial Day was a unique and solemn occasion with blue-clad veterans of the Grand Army of the Republic marching in grave procession. Deaths, funerals, devastating illnesses, ominous quarantines, accidents and drownings were seen at close range and formed indelible pictures. There was the con-



ARNOLD GESELL, M.D.

Portrait by Raymond P. R. Neilson (1950), hanging in
The Gesell Institute of Child Development, New Haven, Connecticut

demned murderer who stared through the bars of the county prison; also the watchdog that was poisoned and the burglary committed in the blackness of the night to invite conjectures and youthful imaginings.

"Strange and sobering things kept happening as if they were part of the normal course of events," Dr. Gesell says in one of his writings. "None of these experiences was overpowering but cumulatively they left a deposit of impressions which sensitized a background for my clinical studies in later life."

The natural course of events took Gesell to normal school at an early age. His mother had won a reputation for successfully teaching a difficult elementary school while in her teens. His father placed a high premium on education and the principal of the local high school, the top of the village's social and cultural hierarchy, was a frequent visitor in the Gesell home. Altogether, it would have been strange had the boy elected to follow a career other than teaching.

On graduating from normal school he taught at Stevens Point High School where his duties included teaching United States and ancient history, German, accounting and commercial geography. He also coached and refereed football. "I do not recall any significant intellectual achievements during that period," Gesell comments whimsically.

With the assistance of a tuition scholarship Gesell attended Clark University where he received a Ph.D. degree in 1906 and an honorary Sc.D. in 1940.

The acknowledged genius at Clark University during Gesell's student days was G. Stanley Hall, whose exciting teachings and magnetic personality helped to mold Gesell's thinking. Dr. Gesell recalls that Hall had "an emphatic propensity to revive within himself the thought processes and the feelings of other thinkers, that this trait enabled him to penetrate the mental life of children, of defectives, primitive peoples and even of animals and of extinct stages of evolution."

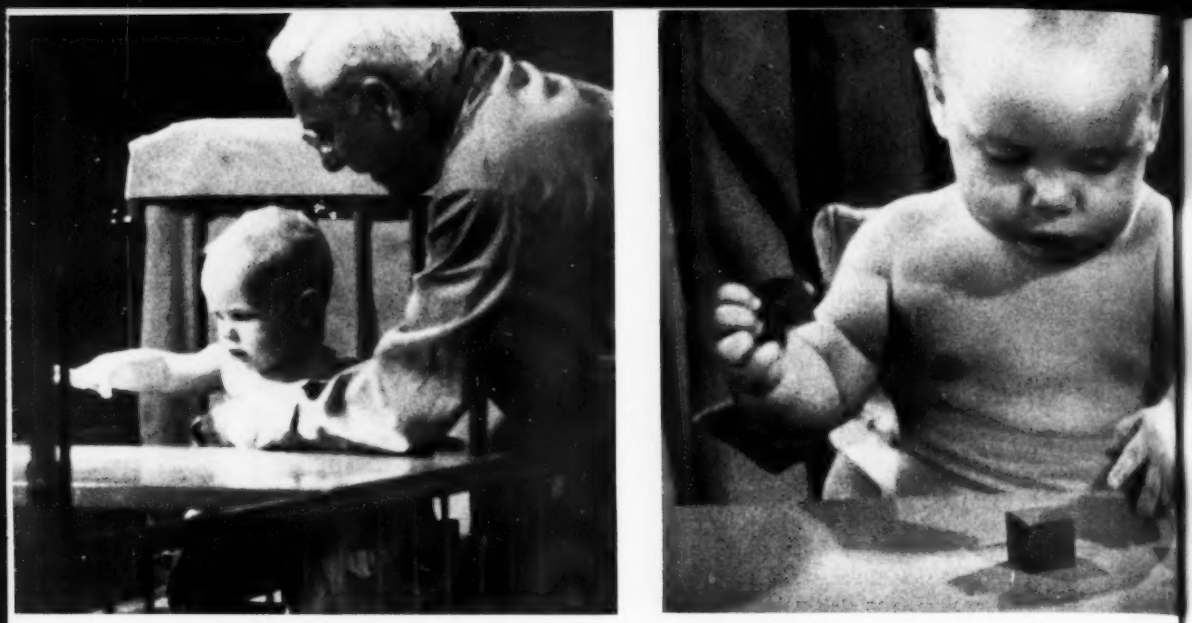
At any rate, the exuberant mind of Hall fired Gesell's imagination and started him thinking along lines which for that day were revolutionary. But many years passed before Gesell accomplished all of the things which had been stimulated by Hall.

On leaving Clark University, Gesell in fairly rapid succession became a counsellor in a boys' camp, an elementary school teacher and an instructor in psychology. It was in the following assignment, as professor of psychology at Los Angeles State Normal School, that his career became anchored in the harbor of its destiny.

While in Los Angeles the young professor became interested in Binet's pioneering efforts to measure intelligence. As a result of this interest, Gesell spent part of the summer of 1909 at the University of Pennsylvania where Dr. Lightner Witmer and his associates were making noteworthy contributions to child welfare. During the same summer Dr. Gesell spent a few weeks at the Training School at Vineland, New Jersey, where Dr. Henry Herbert Goddard, Director of Research, was adapting Binet's principles to a research program. Later, through summer sessions at New York University, he and Dr. Goddard worked together training special class teachers in the care of backward and defective children.

Dr. Gesell has had a lifelong interest in the psychology and education of handicapped children but his mind turned early toward the study of normal childhood. He realized that to understand the nature and the needs of the total organism he must know more about the anatomy, the physiology and the clinical aspects of life and growth. Although he had a doctorate in psychology, he was determined to study medicine. This decision had a far-reaching effect on his subsequent career, because thereby he has been able to demonstrate that there is potentially a clinical science of child development which has important implications in pediatrics, psychiatry and neurology.

In 1910 he embarked on his medical studies, spending a year at the University of Wisconsin in the fields of anatomy and histology. In 1911 he was appointed to a part-time assistant professorship in education at Yale University. This arrangement enabled him to complete his courses and secure a medical degree in 1915.



Dr. Gesell with an infant who has been placed in the clinical crib for a developmental behavior examination.

(From motion picture, "The Embryology of Human Behavior.)

Mrs. Gesell (the former Beatrice Chandler) lent full moral support to this somewhat strenuous educational venture. She had graduated from the University of Chicago at a time when pioneering progressive education was part of the very air the students breathed. Her liberal and creative outlook on child life is reflected in a joint wife and husband book entitled "The Normal Child and Primary Education."

With the approval of Dean George Blumer, Dr. Gesell started a juvenile psycho-clinic in the New Haven Dispensary almost immediately after his arrival in 1911. This was the beginning of the Yale Clinic of Child Development, which with the support of Rockefeller and Carnegie funds launched a program of cooperative research and a diversified clinical service for the diagnosis of infant and child development. Dr. Gesell served as director until 1948. In 1930 he became a member of the Board of Permanent Officers of the Yale University School of Medicine.

The years following the first world war were formative. Dr. Gesell was promoted to a full professorship in the graduate school on completing his medical studies and he coordinated teaching activities with his duties as school psychologist for the Connecticut State Board of Education. In the latter post, the first of its kind created, Dr. Gesell traveled often by horse and buggy from one rural school to another. He identified handicapped pupils and set up individualized programs for their further instruction.

In 1919 he became a member of the Connecticut Commission on Child Welfare. Here his activities contributed to a two volume report dealing with the status of handicapped children together with a formulation of administrative and legislative recommendations in their behalf. The report led to the establishment of the Division for Educationally Exceptional Children which continues to operate under the Connecticut State Board of Education.

Other activities included a mental survey of the elementary schools in New Haven and a study of the vocational capacities of subnormal girls in factory work. In addition, Dr. Gesell examined children of various ages by appointment and studied such special clinical conditions as cretinism, hemihypertrophy, cerebral palsy, deafness, blindness and pubertas praecox. The physical and mental correspondency of a pair of highly gifted twins was investigated. Another pair of twins was studied in detail by the experimental method

of co-twin control over a period from infancy to adolescence.

As Dr. Gesell worked and studied he became more and more aware of the significance of careful observations during the early years of life and he became certain that the developmental patterns of infancy and the preschool years are touchstones to later behavior. And he held that every child manifests a distinctive pattern of development and that the developmental morphology of behavior is therefore basic for constitutional medicine.

Proceeding from this premise he discovered that the vast majority of defects and deviations are present at birth or in early infancy. Thus they can and should be diagnosed during the first year of life. Dr. Gesell found this true in primary amentias, aplasias, malformations and primary degenerations of the nervous system. It was true also in defects and deficits secondary to destructive lesions of the fetal, circumnatal or later periods. It was equally true in the defects brought about by prematurity, endocrine disorders, sensory and motor handicaps, symptomatic retardations and deformations due to abnormal experiences, environmental deprivations and personality defects and disorders. Early diagnoses were possible in these conditions. Upon early diagnoses depended whether or not the children could derive maximum benefits from remedial treatment.

Soon Dr. Gesell came to consider the practice of pediatrics as embracing all those principles and procedures that preserve an integrated approach to the total welfare of the child. This was a far cry from the commonly held view that pediatrics was concerned primarily with feeding formulas and with sick children.

Keen observations and analyses convinced him that whereas a man may be as old as his arteries, an infant is as old as his behavior. Behavior exemplified in sleep, rest, exercise, play, weaning, bladder and bowel control, habits of self care and the so-called behavior problems of thumb sucking, head banging, tantrums and phobias all hold diagnostic importance in terms of developmental health and well-being. In other words, behavior is the master key in the diagnosis and interpretation of the developmental status, whether the physician deals with normal, abnormal or atypical babies.

To convey this concept, Dr. Gesell brought pairs of normal babies of disparate ages into the clinical amphitheater at Yale and demonstrated the influence of development on comparable behavior patterns. And medical students, probably for the first time, observed normality as a clinical entity, accessible to developmental diagnosis.

From the resulting studies and their logical clinical applications came the internationally recognized volume, "The Mental Growth of the Pre-School Child, a Psychological Outline of Normal Development from Birth to the Sixth Year, Including a System of Developmental Diagnosis." This book was recognized as one of the 37 most notable books of the year 1925 and was listed by the American Library Association for the Committee on International Intellectual Cooperation of the League of Nations.

The increasing number of clinical examinations, the cumulative observations of children attending the guidance nursery, now a part of the Yale University Clinic of Child Development, together with periodic contacts with parents and with children of elementary school age led to a further understanding of child development and to further books. Among them were such recognized reference volumes as "Infant and Child in the Culture of Today," "The Child from Five to Ten," "Studies in Child Development," "How a Baby Grows," "Developmental Diagnosis," "The Embryology of Behavior" and a comprehensive two volume "Atlas of Infant Behavior." Collaborating or associated with Dr. Gesell in producing some of these books were such able assistants as Drs. Catherine S. Amatruda and Frances L. Ilg, Henry Halverson, Ph.D., Helen Thompson, Ph.D., Louise Barnes, Ph.D., B. M. Castner, Ph.D. and Janet Learned, Ph.D.

Dr. Gesell's name became a household word in homes and colleges throughout the United States. On the one hand he aided physicians to evaluate abnormal tendencies in infants.

On the other his concise, easily understood writings awakened parents to an awareness of the importance of growth trends in their children. With increased awareness of the natural patterns and tendencies of behavior at progressive age levels, the parents suffered less from unwarranted anxiety and injudicious discipline.

"The success of child care does not depend so much upon rule of thumb technic as upon the underlying attitudes of parents and attendants," he said. "In almost a literal sense the physician can reach the minds of infants and children by altering undesirable attitudes in the parents."

Early in his studies Dr. Gesell became interested in the movie camera as a basic research tool, both to document and to analyze the forms and sequences of behavior patterns. Over the years more than 300,000 feet of film have been assembled and indexed as a photographic research library. Many films have been analyzed frame by frame. Among the important photographic contributions is a motion picture entitled "The Embryology of Human Behavior." This was prepared in cooperation with a team of technical experts interested in producing a conceptual film suitable for medical and other advanced students. The film is based on the work of the Yale Clinic and is narrated by Dr. Gesell. The project was completed under the joint auspices of the Office of Naval Research and the Association of American Medical Colleges.

Yale University became recognized as a leading child development center and became the first institution to accord departmental status in a medical school to the field of child development. Clinically the new science was defined by Dr. Gesell as "a branch of biology which is concerned with the diagnosis and supervision of early human growth in its physical, mental and cultural manifestations."

In 1935 the American Board of Pediatrics set up the field of Growth and Development as a basic requirement for specialty certification. This reflected an increasing emphasis on Developmental Pediatrics as a preventive and supervisory division of clinical medicine. Dr. Gesell is a fellow of the American Academy of Pediatrics and a member of the National Academy of Sciences. Recently he was made president of the American Academy for Cerebral Palsy.

Although Dr. Gesell was retired as Professor of Child Hygiene and Director of the Yale Clinic of Child Development on July 1, 1948, this eminent authority continued his research for two more years as head of a child vision research project. This has resulted in a collaborative volume, "Vision: Its Development in Infant and Child," setting forth a systematic approach to "developmental optics concerned with the ontogenesis and organization of visual functions in their dynamic relation to the total action system."

Dr. Gesell was appointed to a national Sigma Xi lectureship in 1949. He also served as a research associate in the Harvard Pediatric Study.

The advancing years have added to Dr. Gesell's distinguished appearance as well as to his fund of knowledge. There is seemingly no reduction in his magnificent strength and vitality. His blue eyes twinkle as he talks of his five grandchildren and of his plans for the future.

"I have not experienced any loss of interest in the doctrine of development since my chronologic retirement," he says. "There are too many fields of endeavor in education, in child care and in child guidance where a developmental philosophy and a developmental approach are badly needed, both for child and for adult."

With attractive modesty Dr. Gesell points out that science has but started to plumb the depths of the child mind. He says, "Our present knowledge of the child's mind is comparable to a fifteenth century map of the world—a mixture of truth and error, with the heads of strange sea monsters ominously rising out of the dark depths of uncharted seas. Vast areas remain to be explored. Much of our knowledge is disjointed and topical. Many current ideas are sheer speculative derivations rather than the fruit of observation. There are scattered islands of solid, dependable fact, uncoordinated with unknown continents."

MIRIAM ZELLER GROSS

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Letters from Abroad

FROM POSTGRADUATE MEDICINE'S CORRESPONDENTS

PARIS

Treatment of acne vulgaris and of acne rosacea—We in France are in general agreement with Day and Anderson* as to the importance of *local treatment* of acne vulgaris, whether it be a question of extirpating comedones or of evacuating pustules, for the essential objective is that of "liquidating the past." In order to attain this end, some dermatologists prefer incision to scarification, followed by manual expression and peeling (after a precautionary patch test) with Unna's resorcinol ointment.

Except for the milder forms of the disease, only a generalized therapy carries the promise of a permanent cure. Many doctors have lost faith in the efficacy of vitamins or dietary regimens. Recent publications differentiate sharply between acne vulgaris and its treatment by nondietary measures, and acne rosacea for which dietary measures are in order (omission of coarse, spicy foods and of bulky meals), supplemented with hydrochloride and pepsin medication. In fact, treatment of acne vulgaris by gonadotrophic hormones and of acne rosacea by means of certain antibiotics constitutes the therapies essentially recommended here at present (Aron-Brunetiere):

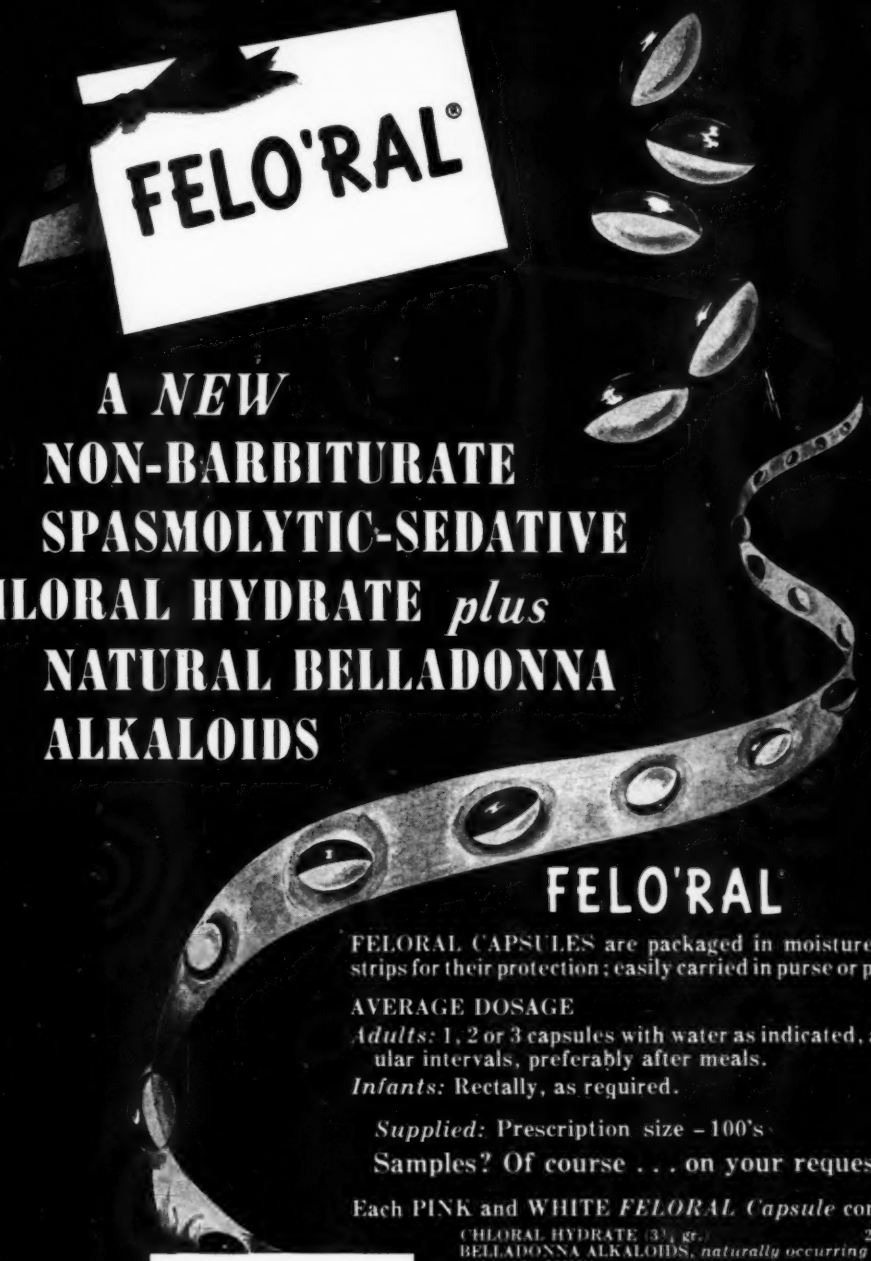
1. Acne vulgaris—The androgen-estrogen imbalance which is the cause of acne is itself apparently due to an imbalance in the gonadotrophic-pituitary relationship, FSH/LH. In fact, the chorionic gonadotrophic-lutein stimulation aggravates the spread of acne. Although gonadotrophin is principally follicle-stimulating, it also exerts a lutein-stimulating effect when administered in large doses. Intramuscular therapy with this hormone in the usual dosage has consequently been productive of a large number of failures. Attempts have therefore been made to produce only the follicle-stimulating effect of equine gonadotrophin and to make this effect as long-acting as possible. These objectives apparently have been obtained successfully by making use of pellet implants containing the low hormone dosages of 1000 units for treatment of men and 2000 units for women. The implants are renewed at intervals of 20 to 25 days, with three to five renewals being required for a cure.

The successful cures which have been obtained in this way in equal measure in both sexes (85 to 90 per cent) are superior to what has been realized with the estrogens. Improvement in menstrual disturbances is simultaneously brought about in the majority of the women treated; constipation, if present, likewise disappears. Such failures as occur seem to be due to antihormones. No untoward reactions were experienced, either local or general, with this therapy.

2. Acne rosacea—This condition seems to be an allergic manifestation in which the allergen is most often identical with saprophytic bacteria which do not produce local infective symptoms. The use of antibiotics is therefore indicated. Since the trouble probably is caused by intestinal infestation *(Salmonella)*, chloramphenicol is the therapeutic agent of choice (6 gm. in eight days). It is successful in about 65 per cent of the cases. Some of the patients who do not respond to this antibiotic do so to AUREOMYCIN® administered in the same dosage. The response seems to indicate that the bacteria are of the gram-positive kind. Some other patients seem to respond only to the successive application of both these antibiotics. Two or three courses of treatment (3 gm. in four days) are frequently necessary to effect complete cures.

(Continued on page A-20)

*Day, R. L. and Anderson, N. P.: The management of acne vulgaris. Postgrad. Med. 12:34-40 (July) 1952.



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MADRID

New Granada hospital—During the ceremonies on October 12 commemorating the discovery of America, Generalissimo Franco dedicated three new buildings of the Hospital of the Medical School of Granada, including the nursing department for specialties, the outpatient department and the building for general services. The specialties building contains the Cancer Institute, supported by the St. Francis and St. Candide Foundation and established by a bequest of 15 million pesetas from Doña M. J. Castillo. The building housing the outpatient department has laboratories, x-ray, diagnostic, therapy, emergency and physical therapy departments, and 10 clinics, each with several examining rooms.

National congress on pediatrics—Important conclusions reached at this congress which was held in Barcelona in October were:

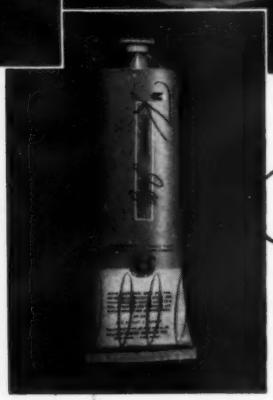
Leishmaniasis—The compulsory declaration is recommended. More information for rural doctors, on symptoms and diagnosis, is needed, as well as a sufficient supply of adequate drugs.

Children's growth—In order to obtain normal standards for Spanish children, it is urgent that standards be established for measurements and technics.

Medical control of teachers—There must be actual regulations on all kinds of teachers. The School Medical Service must be expanded.

Infant mortality—It was recommended that special centers be set up for premature and congenitally weak babies, as a measure of control of infant mortality.

Children deprived of normal family—Lawmakers and sociologists should study the possibility of withdrawing parental authority from persons who abandon their children. There is a need for adoption and home placement centers.



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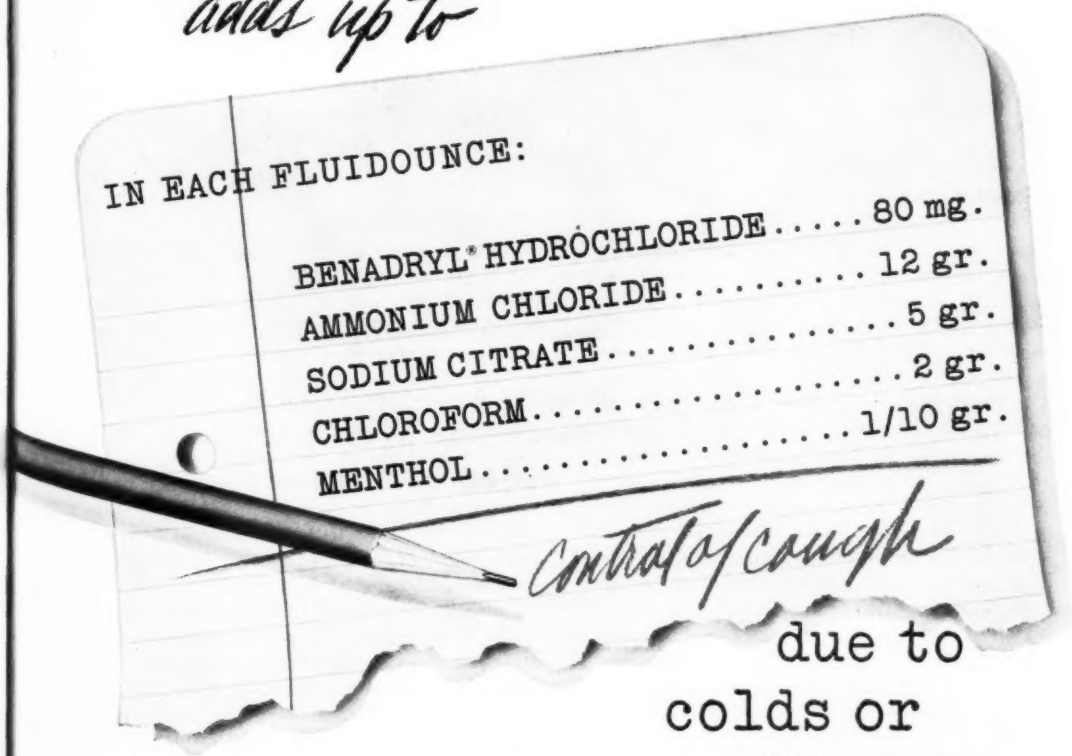
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• TETANUS IMMUNIZATION SCHEDULE FOLLOWING INJURY

Question: 1. What kind of tetanus protection (antitoxin or toxoid) would you give to a patient at the time of an accident who was previously active immunized by the use of triple vaccine followed by one or several booster injections?

2. What is the importance of the time elapsed between the last booster and the accident? When should one use the booster injection? When the antitoxin? When both simultaneously?

Question 2 is the subject of great controversy as far as I know. Some authorities feel that antitoxin has to be given if there elapsed more than one or two years after the last booster; some have five years as a limit for the use of toxoid at the time of an accident (e.g., "Current Therapy 1952"). Some never use antitoxin if the patient has been previously active immunized. Could you help me clarify this important problem?

M.D.—Delaware

ANSWER: 1. In the case of an individual who had received basic immunization against tetanus with tetanus toxoid or a triple vaccine containing tetanus toxoid within five years or, following such basic immunization, a booster injection within five years prior to an injury involving a risk of tetanus, a reinjection of tetanus toxoid at the time of injury should provide adequate protection (see references).

2. The importance of the time elapsed between the last booster and the accident is that there should be sufficient basic immunity so that a booster dose of toxoid will provide a protective antitoxin titer within the incubation period of tetanus. It is not necessary that the individual have a protective level of antitoxin at the time of injury; it is only necessary that the recall mechanism be activated. As stated in the reply to question 1, there is adequate evidence that a booster dose provides adequate protection, if injury occurs not more than five years after previous immunization. There is some difference of opinion as to what should be done in case of an injury taking place at a later date. Your consultant is of the personal opinion that basic immunity would probably result from a booster dose even after a much longer interval than five years but, as far as he knows, this is not supported by actual experimental evidence based on antitoxin titrations. Nevertheless it is his opinion that the risk to the patient would not be greater than that involved in the administration of tetanus antitoxin in view of the complications which have followed the use of that product.

When the interval is greater than five years, tetanus antitoxin and tetanus toxoid may be administered simultaneously if it is felt that antitoxin is required in addition to the toxoid. Repeated doses of antitoxin will thus be avoided, as active immunity will develop before the passive immunity from the antitoxin has been lost. Passive immunization with a prophylactic dose (1500 units) of tetanus antitoxin is, of course, required when there has been no previous immunization. This dose must be repeated at intervals of 7 to 10 days if conditions predisposing to tetanus continue to exist, and especially if surgery must be done.

W. G. WORKMAN

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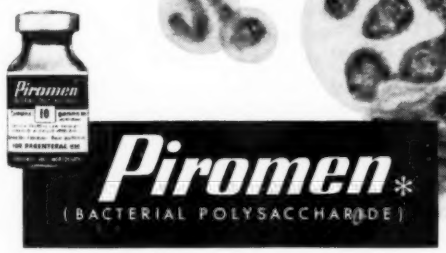
(Continued on page A-24)



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• EXCESSIVE SWEATING AND ENLARGED PORES

Question: Does excessive perspiration cause permanent changes in the skin such as hypertrophy of sweat glands? One of my patients is a laundry worker. She is constantly exposed to high temperature and humidity and she has large pores on her forehead and nose. Are astringents helpful?

M.D.—Missouri

ANSWER: There is no proof that excessive perspiration causes any permanent skin changes. Hypertrophy of the sweat glands has not been noted in individuals after prolonged sweating, as observed in stokers in the holds of ships and others exposed to excessive heat. Large pores are due to the prolonged stretching and loss of tone of the arrectores pilorum muscles of the follicles. This is occasioned by plugging of the hair follicles either by comedones or hyperkeratinization. When the muscles surrounding the follicular openings have lost their tone, astringents are of no value.

BEDFORD SHELMIRE

• NARCOTICS AND IMPOTENCE

Question: A 35 year old man, the father of three children, now complains of impotence. He received narcotics—morphine and DEMEROL®—for about two months following a traumatic injury. He has been off of narcotics for about one month. Do narcotics given over such a length of time usually cause impotence, and is the reaction reversible following the withdrawal of the narcotic?

M.D.—New York

ANSWER: Addiction to morphine regularly leads to loss of libido in both sexes. Potency is not lost during maintained addiction in the male. The length of time required to obtain an orgasm is greatly increased. The condition is readily reversible following withdrawal of morphine and no permanent physiologic or anatomic damage to the gonads results from addiction to morphine.

With respect to addiction to demerol, no information is available on these questions. In all probability the situation in demerol addiction is identical to that during morphine addiction. It seems quite probable, therefore, that the impotence in the patient is due to causes other than his addiction.

HARRIS ISBELL

• STERILIZATION OF KNIFE BLADES

Question: Please advise as to the accepted procedure for sterilization of knife blades and blood lancets, with particular reference to prevention of infectious hepatitis.

M.D.—Virginia

ANSWER: The accepted procedures for the sterilization of knife blades have been modified from time to time. One of the chief concerns, in addition to adequate sterilization, was the preservation of the cutting edge. For this reason, oil sterilization was used for a number of years. With the present day widespread use of detachable blades, the problem of avoiding infectious hepatitis is a relatively unimportant one. If blades are to be used more than once, the same type of sterilization of the blades should be used as for lancets, needles and syringes. Heat sterilization should be (1) by autoclaving for 15 minutes at 121.5°C., (2) by dry heat, for two hours at 170°C. or (3) by boiling in water for 15 minutes at 100°C. If a cold preparation is wished for knife blades, they can be soaked in formalin alcohol solution 17 hours before using.

RICHARD B. CATTELL

• KELOID REMOVAL IN CESAREAN SCAR

Question: A 30 year old white woman has a keloid in a cesarean scar. The operation was done almost two years ago. The scar is about 6 inches long, ½ inch wide and is elevated ¼ inch. It is becoming more and more painful and is perhaps increasing in size.

What is the procedure of choice for its removal? The patient isn't at all keen about more surgery. Is any nonsurgical treatment of value?

M.D.—Ohio

ANSWER: The opinion in regard to the treatment of keloid differs somewhat, and it may be possible to remove the keloid surgically. However, they tend to recur.

Recently a good many of these patients have been treated by radium. X-ray has been used. With radium treatment most of the keloids will grow smaller and lose a good deal or all of the painful manifestations. I think radium is well worth a trial.

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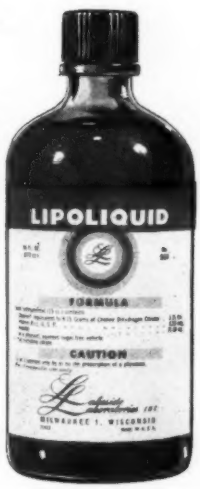

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RHEUMATIC FEVER*

PERTINENT advances in our knowledge of rheumatic fever were reviewed in a series of 23 papers given by recognized workers in the special fields of bacteriology, immunology, pathology, clinical medicine, cardiac surgery and related specialties during a three day symposium held at the University of Minnesota late in 1951. Through the cooperative efforts of the Continuation Center and the Minnesota Heart Association it was possible to attract outstanding workers in each of the major fields of interest in the wide area of rheumatic fever. Sessions usually concluded with short discussion periods that often brought out some significant material not included in the papers presented.

The first session was devoted to the epidemiology and inflammatory changes following rheumatic fever. Current concepts of the frequency and epidemiology of rheumatic fever were neatly summarized by Drs. T. Duckett Jones and Francis Schwentker. A most interesting contribution was the detailed description of the Aschoff body, its modification when observed in the various tissues of the body and the factors concerned in its production, drawn largely from his own experience at the Rockefeller Institute by Dr. George Murphy. Dr. Thomas Dry concluded the session with a review of the congenital heart diseases with inflammatory lesions.

The second session confined itself to the problems of infection and spread of the disease. The detailed epidemiologic studies of Rammelkamp, Denny and Wannamaker at the Warren Air Force Base in Wyoming and the careful studies of Rammelkamp in Cleveland indicate that bacterial factors are important, once a streptococcal infection has been established. The subsequent paper by Rantz, Maroney and Di Caprio on a four year study of newborn infants and children attending the outpatient clinics of Stanford University substantiated the finding that the rate of infection remains about the same in all groups, though the reinfection rate decreases with time and with age. The essential difference between rheumatic and nonrheumatic myocarditis was the lack of involvement of the endocardium and pericardium in most cases of nonrheumatic infections; the insult to the myocardium was generally limited to the single attack of the nonrheumatic infection which was as a rule not progressive, whereas the rheumatic infection was slowly progressive.

The third and fourth sessions were devoted entirely to the changes of the tissues and the blood, plasma and other body fluids after an infection with rheumatic fever. Much of the present knowledge of cellular and circulating fluid changes after any infection were reviewed in fairly close detail in this series of papers. This is perhaps of greater interest to the research worker than to the student of clinical medicine.

The last session was of great interest to the practitioner and the clinician. The present status of mitral valve surgery was thoughtfully outlined by Drs. Varco and Baronofsky. Some of the common troublesome and complex problems concerned with the identification of organisms causing subacute bacterial endocarditis were explained by Dr. Fordyce Heilman with usual clarity. The second part of the paper was a review of the clinical aspects of this disease in which the statements on the choice and use of antibiotics are the reflection of the wide experience and sound judgment of Dr. Herrell. The discussion on cortisone and ACTH in the treatment of rheumatic fever by Dr. Hench and associates stressed the importance of giving these hormones early in the acute attack and continuing them until the signs of activity have subsided. The need for individual dosage sched-

(Continued on page A-52)

*A symposium held at the University of Minnesota, November 29-December 1, 1951. Edited by Lewis Thomas, M.D. 349 pages, illustrated. 1952. University of Minnesota Press, Minneapolis. $10.00.

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ules is always an important aspect of this type of treatment.

Dr. Rammelkamp's paper on the prevention of rheumatic fever was based largely on his own observations among the cases he studied at Cleveland and at the Warren Air Force Base. He pointed out the advantages of penicillin preventive therapy for patients who have had attacks of rheumatic fever, and compared the subsequent attack rates with those for patients without penicillin treatment. From these studies and others it seems advisable to consider penicillin and the other antibiotics as valuable agents in the prevention of recurrence of rheumatic fever, through their effectiveness in overcoming streptococcal infections.

The book is a timely review of the newer knowledge on rheumatic fever, and physicians interested in this disease will find many useful suggestions. The entire subject is covered remarkably well and the large bibliography at the end is a guide to further reading. It is regretted that an index was not prepared by the editor for this otherwise excellent book.

H. C. L.

► PSYCHIATRY AND MEDICINE

By Leslie A. Osborn, M.D., Professor of Psychiatry and Acting Director, The Wisconsin Psychiatric Institute, University of Wisconsin Medical School. 494 pages. 1952, McGraw-Hill Book Company, Inc., New York, Toronto & London. $7.50.

Dr. Osborn started as a general practitioner in Australia and the United States. He became a psychiatrist and later a teacher of psychiatry in a medical school. His background of general practice should have enabled him to write a book of interest to nonpsychiatrists and easy for them to read.

Because of the author's origins in general medicine the reviewer hoped to find simple speech and little of that jargon which is so loved by the average psychiatrist. When will psychiatrists learn that if they wish to interest and teach the general practitioner and the internist and other specialists they must use Anglo-Saxon speech, and must write interestingly about the patients whom all of us see?

Actually as the reviewer turned over page after page he ran into paragraphs like the following taken from page 192: "The second group of questions would relate to the probabilities that development along each of these lines would be lovingly fostered by understanding parents, or would be more or less difficult according to circumstances. At the actual time of birth these questions could not have been answered, but subsequent developments afford workable clews. By utilizing hindsight it is often possible to recognize full achievement in some areas, partially unfulfilled tendencies in others, and in certain respects inherent limitations despite favorable opportunity." All of which is, of course, good English, and can be understood if one reads it two or three times.

Interestingly, on page 200, one finds the doctor speaking of "credence given to superstitions about insanity in the family." On page 201 he says again, "Public interest in mental hygiene should be stimulated and sound concepts imparted in place of hurtful superstitions." How are we nonpsychiatrists going to be helped when men who write books on psychiatry apparently have never heard of the fine work of men like Kallmann and many others who have proved, with thousands of statistical figures, that psychoses and neuroses are commonly inherited? Kallmann can now tell a man whose father is schizophrenic or his brother depressed just what the chances are that he too will some day be schizophrenic or depressed.

When such clearcut statistics based on thousands of cases are available, why should our teachers of psychiatry today either completely ignore the subject of heredity or say that it is all superstition or buncombe?

Some psychiatrists, if questioned on this, will say that it is unwise to admit that psychoses are inherited. They say that this discourages patients. But nonpsychiatrists say that in their experience it often greatly encourages a patient, who all his life has feared that he will become insane like his mother or his grandmother, to find that his particular neurosis is his share of the family curse. Many a time such a man has gone out of the office so encouraged to know that he has his share and hence he knows what it is. He does not have to go on having panics of fear when he thinks he too is going insane.

Efforts to teach the average doctor some psychiatry are tremendously needed, but it is doubted that he will take the trouble to read and learn unless the texts are made simple and interesting.

W. C. A.

(Continued on page A-54)

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► MANUAL OF GYNECOLOGY

By E. Stewart Taylor, M.D., Department of Obstetrics and Gynecology, University of Colorado School of Medicine, Denver. 204 pages with 70 illustrations. 1952, Lea & Febiger, Philadelphia. $4.50.

Although this is an excellent outline of the subject of gynecology, a student would also need a textbook of gynecology to have a thorough understanding of the details of this subject.

In chapter 1 the author clearly states the reason for including history-taking in the physical examination. It would be of advantage for him to have stated the patient's marital status. Chapter 2 is an account of the anatomy and embryology of female generative organs. In chapter 3 he outlines the physiology and endocrinology of the female generative tract and describes menstruation and its hormonal action. In this and the next chapter he gives a good discussion of the endometrial glands and the phases of the menstrual cycle, stating the various hormone actions that take place.

Congenital anomalies are clearly described in the next section, and following is a description of the anatomy of the external genitalia plus the diseases which may attack these structures. In chapter 7 the various diseases of the vagina such as *Trichomonas vaginalis* vaginitis and their treatment are outlined. Next come the description of the cervix and the routine treatment of various conditions of the cervix such as childbirth lacerations and polyps. Chapter 9 deals with malignant diseases of the cervix and gives in detail the treatment of diseases of the cervix, including the use of radium therapy with advice in regard to the Wertheim and Taussig operations. Benign conditions of the uterus including uterine fibromyomas, adenomyosis, endometrial hyperplasia, endometrial polyps and endometritis are covered in chapter 10. Chapter 11 is concerned with adenocarcinoma of the endometrium, of which the writer has an excellent knowledge, and its treatment. He describes the technic for detecting carcinoma of the cervix and also brings in the importance of biopsy in this condition.

The next portions of the text are taken up with inflammatory diseases, including various conditions of the ovaries and their treatment as well as benign and malignant ovarian tumors. The subject of endometriosis is dealt with and the treatment is outlined. The importance of conditions in the pelvis as they affect the anatomy of the pelvis following childbirth is described; these include genital lacerations, relaxations, fistulas and displacements. The closing chapters cover the management of menstrual excesses, the various functional conditions involving the pelvis in later life, the question of infertility and the use of hormone preparations in gynecology.

R. D. M.

► HANDBOOK OF FRACTURE TREATMENT

By Edward L. Compere, M.D., Sam W. Banks, M.D. and Clinton L. Compere, M.D., Department of Surgery, Northwestern University Medical School, Chicago. Ed. 3. Illustrated by Harold Laufman, M.D. 424 pages with 223 figures. 1952, The Year Book Publishers, Inc., Chicago. $6.50.

Designed, as previously, to assist the general practitioner and the graduate student, this third edition presents a comprehensive review of the more common fractures and choice of therapy. Aware that other methods are entirely satisfactory for proper end results, the authors offer advice on procedures which according to their personal experience have been found to afford gratifying restoration of function.

This revision has been made necessary by advancements and improvements in the technics of the care of fractures. The use of the intramedullary nail for fixation and the substitution of prosthetic appliances for diseased femoral heads have received such stimulation lately that a report on these features is essential to the knowledge of the practicing physician.

Some chapters have been rewritten and others changed only slightly in factual content, but in each instance descriptive illustrations and radiographic prints are available to lend a visual account of the procedures described.

Briefness and concise evaluations are eminent characteristics. Yet all the more common injuries are discussed and suggestions advanced for proper orthopedic care. This treatise already has stood the test; it should continue to find its way to the perusal of the great majority of practitioners.

H. O'P.

(Continued on page A-56)

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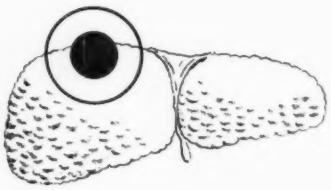

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Books received will be acknowledged in this department each month. As space permits, books of principal interest to our readers will be reviewed more extensively.

Advances in Internal Medicine. Edited by William Dock, M.D., Long Island College of Medicine, Brooklyn, and I. Snapper, M.D., The Mount Sinai Hospital, New York. Vol. 5. 464 pages, illustrated. 1952, The Year Book Publishers, Inc., Chicago. $10.50.

Disorders of the Circulatory System. Edited by Robert L. Craig, M.D. Presented at the Twenty-Fourth Graduate Fortnight of The New York Academy of Medicine, October 8 to 19, 1951. 305 pages with 87 figures. 1952, The Macmillan Company, New York. $5.50.

Rheumatic Diseases. By Eugene F. Traut, M.D., Associate Clinical Professor of Medicine, University of Illinois, Chicago. 942 pages with 192 illustrations. 1952, The C. V. Mosby Company, St. Louis. $20.00.

Malaria. By Paul F. Russell, M.D., Division of Medicine and Public Health, The Rockefeller Foundation. 210 pages with 64 figures. 1952, Charles C Thomas, Springfield, Illinois. $7.75.

Congenital Anomalies of the Heart and Great Vessels. By Maurice A. Schnitker, M.D., Director of Medicine, St. Vincent's Hospital, Toledo. 306 pages with 19 figures. 1952, Oxford University Press, New York. $8.00.

Morbus Alzheimer and Morbus Pick. By Torsten Sjogren, Hakon Sjogren, and Ake G. H. Lindgren, Department of Psychiatry, Caroline Institute, Medical School, University of Stockholm. Translated by Donald Burton. 152 pages, illustrated. 1952, Ejnar Munksgaard, Copenhagen. 25 Swedish k.

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The 1952 Year Book of Pediatrics. Edited by Sydney S. Gellis, M.D., Assistant Professor of Pediatrics, Harvard Medical School. 423 pages with 112 figures. 1952, The Year Book Publishers, Chicago. $5.50.

The White Plague. By Rene and Jean Dubos, Rockefeller Institute for Medical Research. 277 pages. 1952, Little, Brown and Company, Boston. $4.00.

Practical Blood Grouping Methods. By Robert L. Wall, M.D., Department of Research Medicine, Ohio State University Hospital, Columbus. 175 pages. 1952, Charles C Thomas, Springfield, Illinois. $5.00.

Dermatology: Essentials of Diagnosis and Treatment. By Marion B. Sulzberger, M.D., Professor and Chairman, Department of Dermatology and Syphilology, New York University, and Jack Wolf, M.D., Associate Professor of Clinical Dermatology and Syphilology, New York University. Ed. 4, completely revised. 592 pages with 65 figures. 1952, The Year Book Publishers, Inc., Chicago. $10.00.